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ARCHIVES OF SURGERY

VOLUME 19 SEPTEMBER, 1929 NUMBER 3

TRAUMATIC SUBCLAVIAN ARTERIOVENOUS ANEURYSM

FINAL REPORT *

EDGAR LORRINGTON GILCREEST, M.D.

SAN FRANCISCO

It is interesting that a condition for which the treatment is purely surgical should have fascinated one of the world's greatest clinicians, who contributed much to the history and to the present scientific knowledge of arteriovenous aneurysm. No physician or surgeon can fail to be thrilled by reading the contributions of Sir William Osler on this subject. Immediately one is impressed with the true catholicity of the pure scientific mind which he possessed. He himself stated: "Better than any other disease, aneurysm illustrates how borderless are the boundaries of medicine and surgery." There is a wealth of meaning to be gleaned in this one sentence in this day of necessary specialization which frequently leads, unfortunately, to a contracted point of view of disease as a whole.

A study of medical history reveals that the aneurysm has had a particular fascination for many of the greatest masters in medicine. The names which come immediately to mind are Rufus of Ephesus and Antyllus in the second century and, later, William and John Hunter. William Hunter, in 1757, was the first to recognize and to describe the symptoms of arteriovenous aneurysm. John Hunter, a few years later, was the first to operate, and successfully, on a patient with this condition. In the present day, William Halsted and Rudolph Matas have added valuable contributions. Although 1,800 years have rolled by, and indeed, much knowledge, bit by bit, has been acquired, surgeons are still searching for additional information in order to explain definitely and finally two questions in physiology: (1) the exact mechanism of the enlargement of the heart, and (2) the retardation of the pulse rate on closure of the arteriovenous fistula.

The answer to this question of the treatment is likewise sought: whether the ligation of the accompanying vein is imperative in an arteriovenous fistula or at least advisable in a simple aneurysm, unless

* Submitted for publication, March 29, 1929.

* From the University of California Medical School.

* Read before the Southern Surgical Association, Greenbriar Hotel, White Sulphur Springs, W. Va., Dec. 13, 1928.

circumstances permit restoration of the original channels with elimination of the fistula after the method of Matas.

From the point of view of treatment, aneurysms resolve themselves into two types: (1) that in which the artery alone is involved, known as a simple, saccular or fusiform aneurysm, and (2) that in which the artery and vein are both involved and a communication is thereby established between them, known as an arteriovenous fistula or aneurysm. In the latter condition there is more involvement of the venous system, and it is this lesion only with which I am at present concerned. It is most important in the treatment to know definitely which of the two conditions is present.

The multiplicity of the names for this second group of lesions is often misleading. 1. By arteriovenous aneurysm or varicose aneurysm is meant an indirect communication between an artery and a vein by an intermediary sac or channel. 2. By arteriovenous fistula or aneurysmal varix is meant a direct communication or immediate apposition between an artery and a vein.

The report of the present case is of particular interest because of (1) the duration of the arteriovenous aneurysm; (2) the development of the right side of the chest, the right shoulder girdle and the entire upper right extremity to tremendous size, and (3) data collected before and after operation which add important proof to many of the experimental and clinical observations and the studies of Halsted, Matas, Reid and Holman.

The combination of ligations executed in this case, as far as I can ascertain, has never been used before. James E. Thompson, in 1915, ligated the innominate artery successfully for the cure of a subclavian aneurysm in its first portion. He was able to find authentic references to fifty-two similar ligations in which there were sixteen recoveries, that is, 30.7 per cent. Since 1880, the procedure has been followed twenty-six times, with twelve recoveries and fourteen deaths. As Thompson stated, "This death roll, 53.8 per cent, is enormous, but the disease, is almost certainly fatal sooner or later unless treated by surgical means."

A preliminary report of this case was made on Dec. 8, 1927, before the Western Surgical Association in Omaha. At that time, the patient was clinically well. He has since returned to the hospital and additional distal ligations have been successfully accomplished, with the result that all evidence of the aneurysm has disappeared, his arm has decreased to normal size and he "feels perfectly well." I therefore desire to review the case in full and make a final report.

REPORT OF CASE

A Mexican, aged 35, had been married for seven years and had five children. His wife had had no miscarriages. The patient stated that he had always been

well and strong, and that he did not smoke or drink alcoholic beverages. The only accident which he had ever sustained was the following:

On Dec. 12, 1920, while walking down the street in Navojoa, Mexico, about 11 p. m., on the way to his hotel, he was halted by two bandits who were crouched on the ground; the instant that he was ordered to hold up his hands he was shot through the chest. The bandits were in front and somewhat to his left. This gave the bullet an upward and outward direction. Immediately he became unconscious and fell to the ground. After three hours he awoke and found himself in a pool of blood. He gradually made his way to the hotel, about 500 yards distant, resting at frequent intervals. On his arrival there a physician was secured at once.

The wounds were healed in fifteen days. The patient remained in bed about twenty-five days. On the second day after the injury he felt a thrill beneath the right clavicle. The arm was paralyzed and numb immediately after the accident and remained so for four or five weeks. Then there began to be terrific pains and cramps in the entire arm; these gradually lessened. Two or three weeks later the patient was able to move his arm. From the day of injury the arm was swollen and darker than normal. The patient was told by the physician that he had an aneurysm and that he needed an immediate operation. The patient hesitated to have it performed at that time as he desired further consultation.

About Jan. 20, 1921, he returned to his work as bookkeeper. He did not attempt to use his right arm as it was "weak and stiff." He was at this time under no physician's care, and the arm hung limp at his side. He rubbed it frequently to stimulate the circulation. By July, six months after the injury, the pain had subsided a great deal, much strength had returned and there was much better movement of the fingers. The patient now began to work with his right arm and hand. He wrote with it, did bookkeeping and drove an automobile, but he could not use a typewriter or lift any weight. During this time the diseased arm was only slightly larger than the other.

In September, 1921, the patient sought the opinion of a surgeon in Mexico City on account of the increased distention of the veins over the axillary fold and chest. An operation was not advised, and the patient returned home.

From this time until eight months before the first operation, there was little change except that the arm slowly increased in size, and the veins in the axilla and over the anterior part of the chest became more and more distended. During these seven years the patient carried on his regular work about the office and was able to do almost everything except use a typewriter, which was prevented by lack of mobility of the fingers.

About April, 1927, the patient noticed that his entire arm was becoming much larger and heavier, and that he was unable to use it as before. The veins in the upper part of the arm, axilla and chest became more visibly distended. After that time he noticed that if he put his hand down it immediately became much swollen. Also during 1927, he began to have shortness of breath even when standing still in the office. This dyspnea was distressing when he attempted to walk fast or ascend stairs. He found it impossible to run or climb stairs rapidly. He stayed within his reserve and therefore was not compelled to sit down to rest frequently. He never fainted. His sleep was sound and restful. At no time did he have symptoms in the head or notice any swelling of the ankles.

During the six weeks before operation the arm increased more rapidly in size than before. In fact, it became so huge that he found it impossible to insert it in the sleeve of his coat. The arm felt very heavy. The swelling in the region of the elbow made it impossible for him to bend it very much. The swelling in

the hand and fingers prevented any work with the hand. About six weeks before operation the patient also developed two ulcers, one on the dorsum of his hand and the other on the flexor surface of his forearm; these increased in size. As his entire upper right extremity and shoulder had become useless and a burden his physician in Mexico advised him to go to San Francisco and have a shoulder

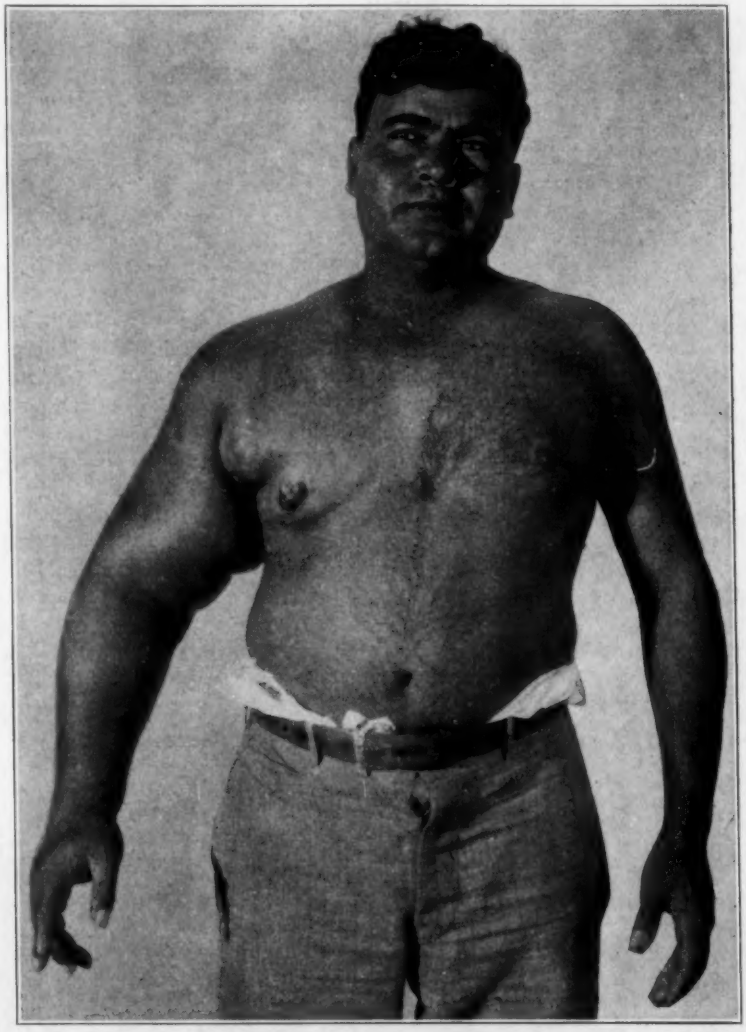

Fig. 1 (Sept. 15, 1927).—Traumatic subclavian arteriovenous aneurysm, six weeks before the first operation. Note the great swelling of the upper arm.

girdle amputation. He reconciled himself to this as inevitable and came to me for the operation, being referred by Dr. Frederick Kroll of San Francisco.

It is interesting that during the preceding seven and one-half years no one suggested elevation of the arm or a pressure bandage to relieve the chronic passive congestion with resulting edematous infiltration. During all this time the valves in the veins were gradually giving away.

Examination.—The patient was a remarkably well developed and well nourished man with an extremely broad chest. There was the scar of a bullet wound of entrance over the third costochondral junction to the right, 6 cm. from the middle of the manubrium, and the corresponding scar of a bullet wound of exit over the posterior aspect of the supraclavicular region, 5 cm. from the neck, and just posterior to the lateral edge of the trapezius muscle; the tract apparently traversed the structures underneath the clavicle at its midportion. There was a tremendous swelling, involving the entire right side of the chest wall (fig. 3) including the breast, the right nipple, which was about 3 cm. lower than the left, the pectoral region, the axillary region, and the entire shoulder girdle, arm, forearm and hand (fig. 4). There were large tumor masses, the size of a grapefruit, consisting of huge dilated veins the size of a finger; these were especially visible in the pectoral and axillary region, in the upper third of the arm and along its flexor surface. Although the patient's skin was naturally dark, it was readily seen that the right hand and arm were several shades darker than the left. This darkening of the skin was conspicuous enough to attract the patient's own attention to it. There were two large, indolent, varicose ulcers about 5 by 7 cm. in diameter on the dorsum of the hand and the flexor surface of the forearm.

Fig. 2.—Six weeks before the first operation. Note the patient's inability to extend the arm fully.

A systolic thrill was distinctly palpable in the supraclavicular region, less distinctly palpable in the infraclavicular region and distinctly palpable in the axillary region and over the lower third of the inner aspect of the upper arm where the basilic vein courses. Here it was like the purring of a cat.

On auscultation over the right supraclavicular region a continuous booming sound was audible, with systolic accentuation. This bruit was distinctly audible but decreased over the right side of the chest. It was again heard distinctly in the axillary region where it sounded like the murmur of machinery. In fact, the sound was similar to the roar one hears in a subway when a train whirls past. Over the lower third of the arm, where the thrill was most palpable, a loud bruit could be heard with systolic intensification. Here the sound was of a much higher pitch and reminded one of the whistling of the wind. This sound was even transmitted to the upper third of the forearm.

Deep digital pressure applied just above the center of the right clavicle made the thrill imperceptible and the bruit inaudible. On release of the pressure, they instantaneously returned. Presumably this pressure completely obliterated the communication between the subclavian artery and vein. It also produced an

immediate elevation in both diastolic and systolic blood pressure, which rose in the left arm from 134 to 154 systolic and from 74 to 94 diastolic; and a retardation of the pulse from 84 to 76, Branham's bradycardiac reaction. Matas stated: "This is by far the most important and early sign of the cardiovascular disturbances brought about by an arteriovenous fistula and is of great diagnostic significance in all cases in which the arteriovenous connection is accessible to compression." The right radial pulse could be palpated with difficulty. The hand felt cold.

There were no disturbances of motion or sensation in the right arm, with the exception that the great swelling made it impossible for the patient to extend his arm or to flex his fingers completely.

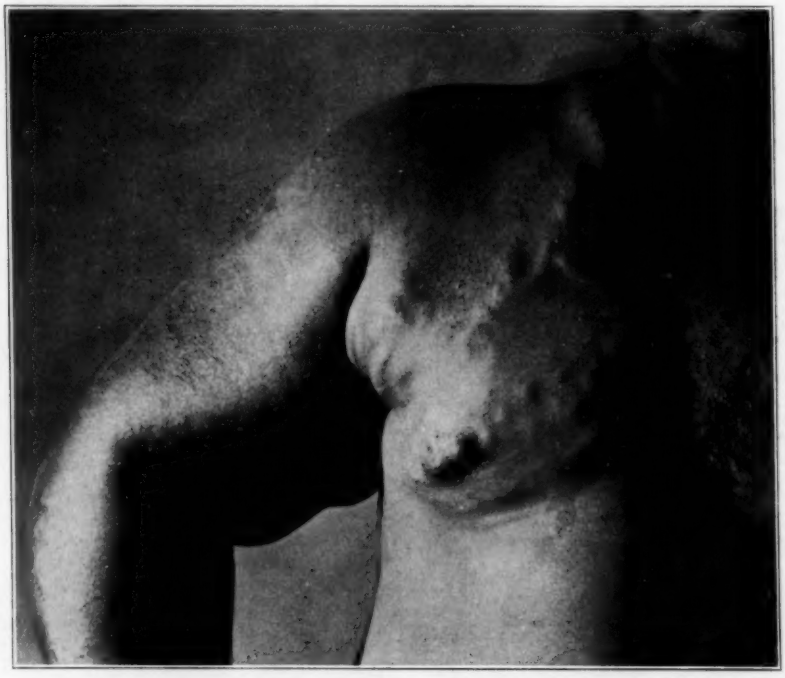

Fig. 3.—Six weeks before the first operation. Note the large lakes of dilated veins over the right side of the chest.

Between the right thumb and index finger the temperature was 35.7 C. and between the left thumb and index finger it was 36.2 C. Examination of the blood showed: hemoglobin, 90 per cent; erythrocytes, 4,800,000; leukocytes, 6,150. The differential count showed: polymorphonuclears, 69 per cent; lymphocytes, 25 per cent; eosinophils, 2 per cent and transitionals 4. The Wassermann test was 4 plus. The urine showed a slight trace of albumin and an occasional hyaline cast.

Blood from a vein of the right arm gave an oxygen content of 11.4 per cent by volume and from the left arm 6.9 per cent by volume; blood from a vein of the right arm gave a carbon dioxide content of 38 per cent by volume, and from the left arm 49.4 per cent by volume.

On Nov. 3, 1927, a 6 foot (1.8 meters) ventral projection showed the heart definitely increased above normal size. The enlargement seemed to affect the left ventricle predominantly. The aorta was neither elongated nor dilated. The right side of the chest was a little less well expanded than the left in its upper portion.

Diagnosis.—The lesion was diagnosed as an arteriovenous aneurysm of the subclavian artery and vein, rather than a simple aneurysm. This conclusion was based on the following factors: (1) the great swelling of the arm, (2) the great dilatation of the veins, (3) the continuous purring thrill, (4) the continuous buzzing or machinery-like murmur accentuated during systole, (5) the centrifugal

Fig. 4.—Six weeks before the first operation. Note the great swelling also of the forearm and hand.

Fig. 5 (Nov. 4, 1927).—One day before the first operation. Note the decrease in swelling after six weeks of elevation and the application of an elastic bandage.

transmission of the bruit and thrill along the course of the axillary and basilic veins, (6) the elevation of blood pressure on temporary deep pressure over the site of the lesion, (7) the retardation of the pulse on temporary deep pressure over the site of the lesion, (8) the increase of oxygen content in the veins of the right arm, (9) the decrease in local temperature and (10) the enlargement of the heart.

For several weeks prior to the operation the patient was kept in bed; his right arm was elevated in a Thomas splint and crepe bandages were applied to the entire arm and shoulder girdle. This proved effectual in decreasing a great deal of the edema. It also afforded the heart a necessary rest (fig. 5).

The plan of operation consisted of the following steps: (1) subperiosteal resection of the proximal two thirds of the clavicle, (2) transection of the muscles of the neck, permitting thereby: (3) the free exposure of the great vessels of the upper part of the chest and neck, (4) possible ligation of the innominate artery and vein and the carotid artery and jugular vein, (5) or ligation of the subclavian artery and vein; and (6) further, if practicable, ligation of the artery and vein distal to, and as close to, the sac as possible, but proximal to the origin of the posterior circumflex and subscapular vessels—in short, a quadruple ligation of the fistula or aneurysm.

This procedure was carried out twice on a cadaver previous to the operation.

The operation was performed on Nov. 5, 1927, at 9:30 a. m. Forty-five minutes before the operation, the patient was given a hypodermic injection of ¼ grain (16 mg.) of morphine and 1/150 grain (0.4 mg.) of atropine. He was placed on the table with shoulders and head slightly elevated; the head was slightly extended and turned to the left side. Local anesthesia of 0.5 per cent procaine hydrochloride was given. This was first injected near the angle of the jaw and just under the chin in the midline in order to apply the towel skin clips without hurting the patient. Starting at a point between the articulation of the clavicle to the manubrium, procaine hydrochloride was injected 3 cm. to the left and over the right clavicle to its outer third and from the manubrium up along the anterior border of the sternomastoid muscle for 7 cm. An incision was now made from the outer third of the right clavicle and slightly below it, and extended up along the anterior border of the right sternomastoid muscle for 8 cm. The skin flap was dissected in both directions. The sternomastoid muscle was divided. The triangular flap of skin and muscle was turned upward and outward. The external jugular vein was ligated.

The sternohyoid and sternothyroid muscles were severed about 1.5 cm. above the sternum and laid back. A subperiosteal resection of the inner two thirds of the clavicle was carried out; it was cut through with a Gigli saw and disarticulated at the sternum. With this exposure the large mass of dilated veins under the clavicle and over the arteriovenous aneurysm, as well as the large vessels of the neck, came clearly into view (fig. 6). The arteriovenous aneurysm involved the second and third portions of the subclavian artery and vein. It should be recalled that the subclavian artery and vein are not companionate in their first part but are separated by the anterior scalene muscle. The mass of large veins over the aneurysm were adherent to each other and to all the surrounding tissues. One of the larger veins, thought to be the subclavian, was ligated at its outer third, as a thrill could be felt over the proximal area.

Gas and ether were administered at 10:30 a. m. The carotid artery was located with the adjacent vagus nerve and dissected down along the anterior and inner surface until the subclavian artery could be felt coming up from behind and bifurcating with it at an acute angle. The subclavian artery was then followed up. It was seen to be enlarged and to emerge into the aneurysm in its second portion. The subclavian vein in nearly its entirety was part of the aneurysm, the remaining proximal end being greatly dilated. For this reason it was decided to ligate the innominate vein. Then the internal jugular was ligated, medium braided silk being used for the ligations. In attempting to push the pleura downward and outward in order to free the subclavian artery, a slight tear occurred in it producing a slight pneumothorax. Contrary to the observation of most authors, the first portion of the subclavian artery was not found difficult to ligate. Heavy braided silk was used and a double ligation was performed after the method of Ballance and Edmunds. With the exposure obtained, one could dis-

tinctly see the best site for the ligation and could feel sure that the recurrent laryngeal nerve was not incorporated in the ligature.

No thrill could be felt now, but as there was still some dilation of the veins over the aneurysmal varix, one of the large veins toward the distal portion was ligated. In attempting to free this large vessel with its thin wall from the adherent tissue and veins, and to pass the aneurysm needle around it some venous hemorrhage occurred which was fairly easily controlled. The mass immediately became smaller with the ligation of this vessel, which was thought to be the subclavian vein. In all, I ligated the innominate vein, the external jugular and the internal

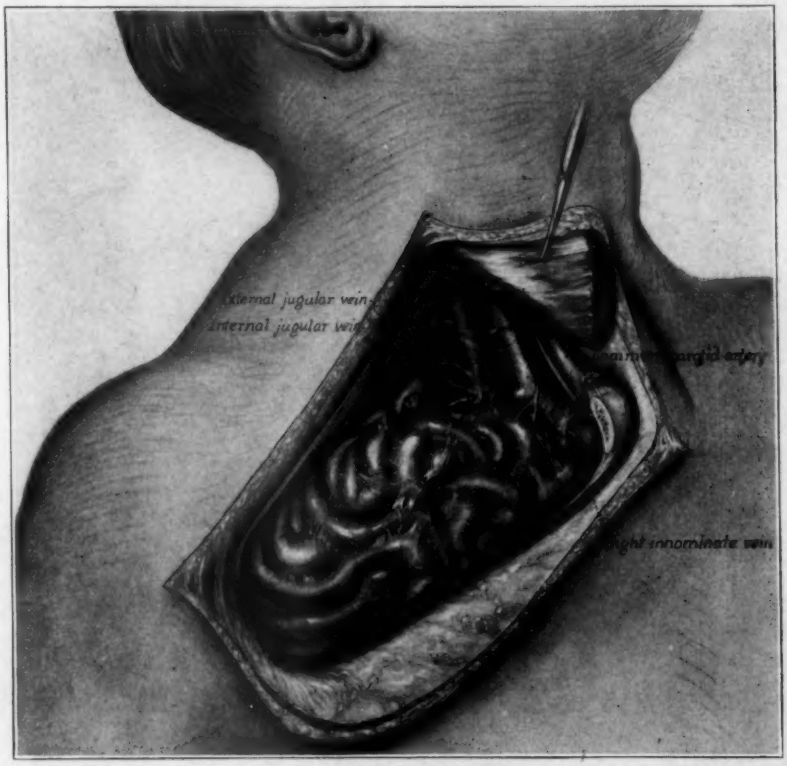

Fig. 6.—Great mass of dilated veins beneath the clavicle. The aneurysm is between the subclavian artery and vein.

jugular veins, the subclavian vein in two places and the subclavian artery in its first portion (fig. 7).

The question that naturally presented itself then was whether to attempt further dissection through this veritable wilderness of matted dilated thin-walled veins and to execute a distal ligation of the artery and vein, that is, the third part of the subclavian artery and vein or the axillary artery and vein with a possibility of dissecting out completely the aneurysmal sac, or to rest on the laurels won. As the procedure had already consumed more than three hours and as no thrill could now be felt nor bruit heard, it was decided to adopt the latter course, in the hope that with the aid of these ligations nature might close the fistula, if any

remained. The severed muscles were brought in apposition and sutured, and the wound was sutured in layers without drainage.

Postoperative Progress.—The patient manifested practically no evidence of shock. The wound healed per primam. Some swelling in the right supraclavicular space was observed on the sixth day, when the stitches were removed. This was due to an accumulation of serum which was aspirated on the eighth, tenth, thirteenth and twenty-first days after the operation. The fluid did not return.

The blood pressure which previous to operation was 134 systolic and 74 diastolic was 136 systolic and 74 diastolic one hour later. The next day, it was 150 systolic

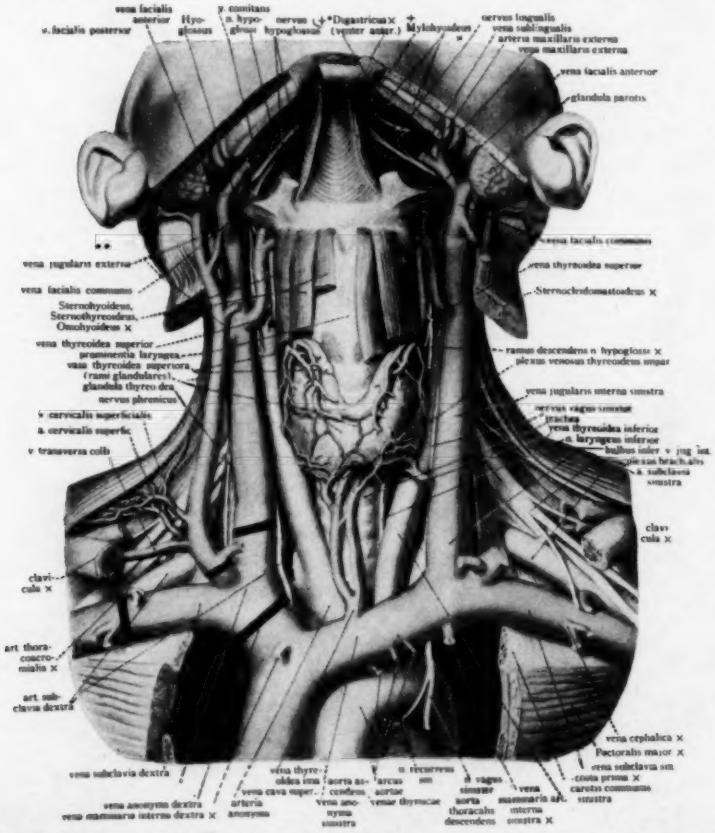

Fig. 7.—Sites of the ligation of the external and internal jugular, subclavian and innominate veins and the subclavian artery. (From Sabota's Anatomy.)

and 112 diastolic; two days later, 152 systolic and 86 diastolic, and the third day, 164 systolic and 82 diastolic; it then gradually declined until on the twenty-eighth day it was 124 systolic and 74 diastolic.

The day following the operation, the patient complained of severe pain in the lower right anterior axillary region when an attempt was made to change his position or to move his right arm. On the tenth day the classic signs of fluid were present in the right lower portion of the chest, and a roentgenogram revealed a fluid line. A thoracentesis was performed on the fifteenth and seventeenth days, but only on the latter day were 2 ounces of sanguineous fluid aspirated. This was repeated on the twentieth day with the same success. Two days later no fluid

could be seen in the chest. The lung, which had been collapsed, due to the pneumothorax, was now expanded. The diaphragm was high and did not move, indicating that the right phrenic nerve had been injured.

A diminution in the size of the right side of the chest, the entire shoulder girdle, arm, forearm and hand was conspicuous after the second day, and continued uninterruptedly, as shown by the accompanying table of circumferential measurements (table 1). This decrease in the venous channels is of particular interest when it is remembered that the innominate, subclavian and jugular veins were ligated.

On Dec. 5, 1927, twenty-eight days after operation, the patient was feeling well. The swelling of the arm was decreasing steadily (fig. 8). The bruit was becoming less and less distinct. Only a slight thrill was palpable. The patient returned to his home in Mexico that day.

Whether in this case a quadruple ligation should have been carried out at this time, thereby subjecting the patient to considerable additional risk, deserves consideration. Time will best answer this question. While the surgeon should be daring he must never lack restraint. If I had performed this operation, there

TABLE 1.—*Circumferential Measurements During the First Stage of Treatment, 1927*

Area	Oct. 20	Oct. 25	Nov. 14	Nov. 19	Nov. 24	Dec. 3
	Cm.	Cm.	Cm.	Cm.	Cm.	Cm.
Wrist	19.25	18	18	17.25	17.0	16.5
Middle forearm, 14 cm. above wrist	32.0	29	26	25.0	25.0	24.5
Elbow	40.0	34	33	32.0	31.0	29.5
Middle arm just below greatest swelling	52.0	43	42	41.0	38.5	36.0
Over greatest swelling, upper third of arm	55.0	51	49	47.0	43.5	41.0
Axilla	53.0	50	47	46.0	45.5	44.0
Around shoulder girdle, 9 cm. from nipple	66.0	61	60	55.0	55.0	54.0
Right side of chest at leval of midline	68.0	64	56	53.0	53.0	52.5
Left side of chest at level of midline	50.0	50	50	50.0	50.0	50.0

would have been no opportunity to see what nature could do in finally closing the fistula or aneurysm when partial ligation had been accomplished. As Halsted said, "Surgeons should bear always in mind the opportunities which they have daily at the operative table to strive for results which may be contributory to the advancement of their science. The operating room is a laboratory for the surgeon."

Second Admission.—On Aug. 6, 1928, the patient was readmitted to the hospital, stating that he had felt perfectly well since the first operation on Nov. 5, 1927. He had been working regularly, using both arms and hands with ease. The great swelling of his shoulder, arm and hand had not returned. Seven weeks before the second admission, however, with the advent of very warm weather, he noticed that the veins in the axilla became larger. Coincidentally, a varicose ulcer developed over the flexor surface of the right forearm. Six days before, he had awakened during the night to find that a considerable hemorrhage had occurred in this ulcer and that he was lying in a pool of blood. During the next five minutes he continued to bleed so profusely, that he thought he would die. Pressure controlled the hemorrhage, however, and he left Mexico the next day for San Francisco.

Examination.—Examination (fig. 9) at this time revealed a slight increase in the swelling of the right shoulder girdle and arm, but there was no return of the

great swelling seen prior to the first operation. The patient was able to slip his arm into his coat sleeve and he walked about with ease. The circumferential measurements are shown in table 2.

Over the flexor surface of the midportion of the forearm there was an ulcer, 3 cm. in diameter, surrounded by markedly discolored skin and an induration of the soft tissue. In fact, the entire right arm and hand were much darker than the left. No pulse could be felt at the right wrist. There was no impairment of motion or sensation at any point. The clavicle, the inner half of which was removed, showed definite regeneration.

Just above this regenerated clavicle, at its midpoint, there was still a thrill on deep palpation. This thrill could also be felt on deep palpation in the axilla over

Fig. 8 (Dec. 5, 1927).—One month after the first operation. Note the great reduction in the swelling of the right side of the chest and the entire right arm.

Fig. 9.—Note the gradual recurrence of the swelling, nine months after the first operation.

the mass of tortuous large veins which lay just underneath the skin. There was a point just above the elbow where the thrill was distinctly felt and heard on auscultation. Deep pressure above the clavicle resulted in a complete disappearance of the thrill and bruit and visible recession in the size of the dilated veins in the axilla. Before the closure of the fistula the blood pressure in the left arm was 140 systolic and 82 diastolic. On closing the fistula the blood pressure immediately rose to 158 systolic and 90 diastolic, dropping as long as the fistula was closed to 150 systolic. Accompanying this increase in blood pressure, there was a slowing of the pulse from 72 to 64. The heart sounds were normal. The apex beat could barely be felt in the nipple line. There was no increase in precordial activity and the heart was not demonstrably enlarged to percussion.

The hemoglobin was 90.5; the red cells, 4,900,000 and the white cells, 9,900. The Wassermann reaction was 3 plus.

Examination of the urine revealed a slight trace of albumin.

On Aug. 8, 1928, the right leaf of the diaphragm was still very high, the dome reaching just above the fifth rib anteriorly. It was not quite so high as on Dec. 1, 1927.

The heart was still large; it appeared appreciably larger than on Nov. 3, 1927, before the first operation. Measurement of the shadow of the heart vessel by planimeter showed that on Nov. 3, 1927, it was 189 sq. cm., and at the time of the second admission it was 198.3 sq. cm. Apparently the high diaphragm produced a torsion of the heart with a greater apparent than actual increase in size

It was quite evident that the condition was an arteriovenous aneurysm which had reestablished itself following ligation of the subclavian artery in its first portion and ligation of the innominate, subclavian and jugular veins about eight months before.

Operation.—It was decided: (1) to resect the completely regenerated inner portion and outer third of the clavicle which was still intact; (2) to expose the axillary vessels beyond the communication between the vessels, which lay probably in the third portion of the subclavian artery; (3) to identify the structures distal to the fistula, and (4) to expose the aneurysm itself, separate the vessels, preserve the artery and vein, if possible, and if not, excise the aneurysm.

Operation was performed on Aug. 10, 1928, at 9 a. m. Gas anesthesia was used throughout the operation. The patient was placed in position with the arm at right angles to the body, and the shoulders slightly elevated. An incision was made along the line of the old scar extending laterally to the coraco-acromial articulation. The regenerated portion of the clavicle was removed as well as about 2.5 inches (6.3 cm.) of the intact clavicle. Numerous dilated veins were encountered in approaching the clavicle. Whether it was due to the position of the arm it is difficult to say, but it was almost impossible to palpate the thrill which previously had seemed quite prominent over the center of the clavicle. There was no pulsation to be felt in any of the vessels except in the common carotid artery on the right side. Identification of structure was extremely difficult, primarily because of this absence of the thrill and pulsation; the scar tissue resulting from the previous operation still further obscured the normal tissue. It was felt wise to continue the dissection down the arm in an endeavor to expose normal tissue and then work back toward the supposed location of the communication. The subclavius muscle was divided as was a considerable portion of the pectoralis major.

After some difficulty the axillary vein was finally identified. This hugely dilated vein had a diameter of 2.5 cm. On further exposure the vein was found to be pulsating; closure of it resulted in the identification of the aneurysmal sac about the size of an English walnut which lay just below and posterior to the middle of the clavicle. A faint thrill could be felt over this aneurysmal sac which was completely controlled by closure of the axillary vein beyond it. Closely adherent to this sac, just superior to the axillary vein were cords of the brachial plexus; these, in turn, surrounded the axillary artery which at this point was approximately 4 mm. in diameter. There was no pulsation to be felt in this axillary artery beyond the aneurysmal sac. This resulted in a considerable delay in its exact identification. The ligation of the axillary vein with a piece of narrow tape produced a conspicuous and easily palpable pulsation of the aneurysmal sac. An attempt was then made to find the arterial connections with this sac, but

pressure at any particular point failed to influence the pulsation greatly. The subclavian artery was identified, lying just posterior to the scalenus anticus muscle (fig. 10).

It is interesting to note that this artery was not palpable before ligation of the axillary vein, primarily because of the large venous reservoir into which it emptied. Closure of the subclavian artery just beyond its emergence from the scalenus muscle resulted in a marked diminution in pulsation, but not in the complete

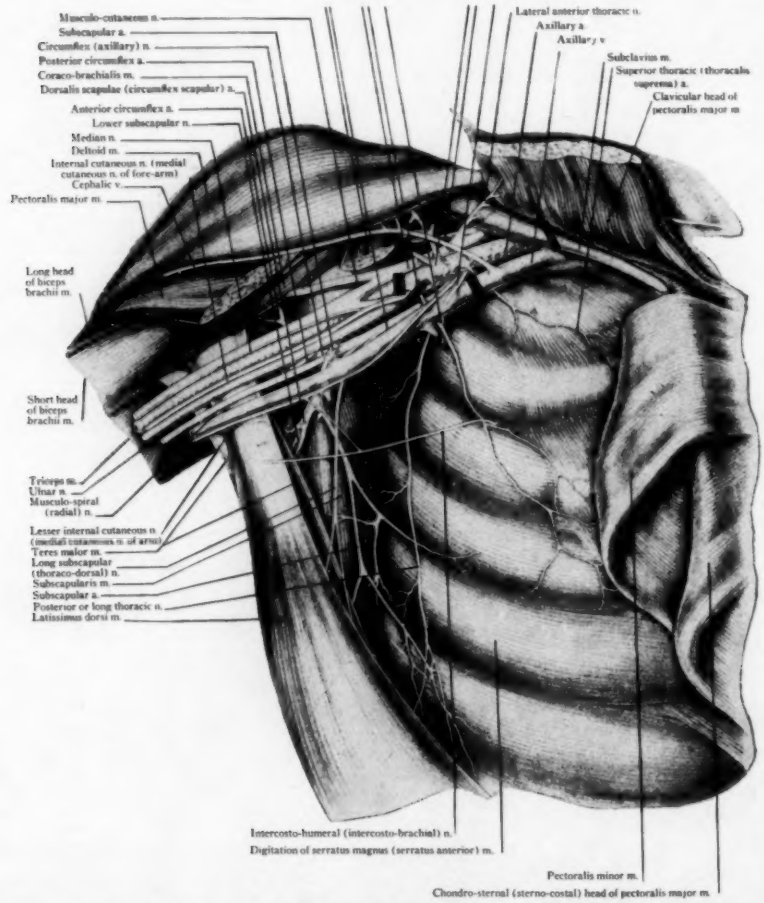

Fig. 10.—Sites of ligation of the axillary and long thoracic veins, and the subclavian, axillary and long thoracic arteries. (From Deaver's Surgical Anatomy.)

control of the arterial supply of the sac. It was ligated with braided silk. The axillary artery was ligated with silk, with a little further diminution of the pulsation of the sac. The long thoracic artery and vein lying posterior to the axilliary vein were also ligated with braided silk. This controlled the pulsation of the sac almost completely. It was felt, however, that with ligation of the axillary vein and almost complete control of arterial supply, the aneurysmal sac would probably be closed by thrombosis. It was thought that the subclavian

artery in its third portion emptied almost completely into this aneurysmal sac which in turn emptied into the very large axillary vein. It is worthy of mention that when the axillary vein was ligated, the general blood pressure rose about 10 points. All bleeding points were controlled during the operation with sutures and ligatures of silk. The cephalic vein was dilated to a diameter of about 1 cm. The subcutaneous tissue and skin were closed with interrupted sutures of silk.

Immediately after the operation the hand which formerly had felt cold now had good color and was warm. There was unquestionably an immediate improvement in the circulation through the procedure followed.

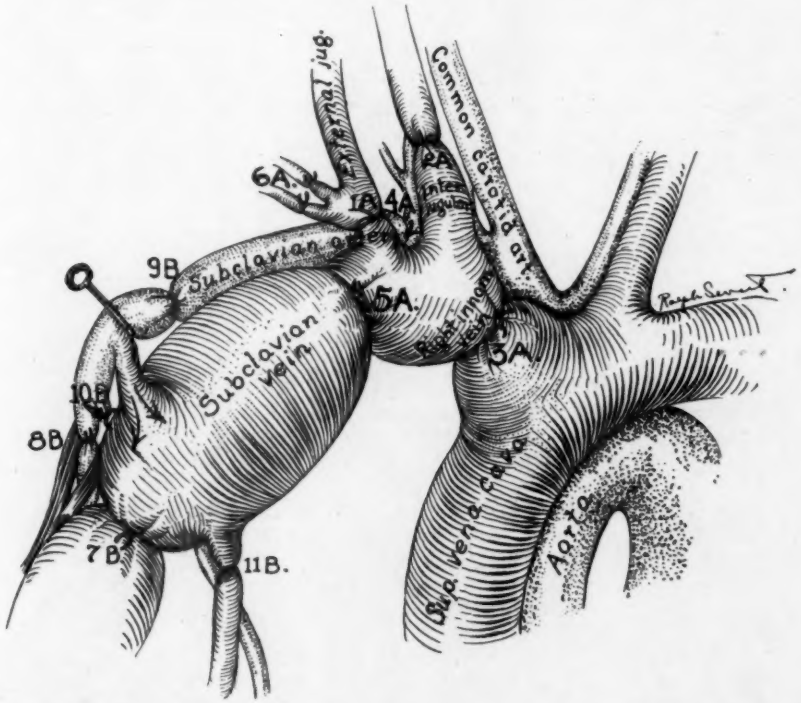

Fig. 11.—The operation as completed in two stages. The ligatures are numbered in the order of their application.

The great difficulty encountered in the second operation, which was tedious and required six hours, was further proof that it would have been inadvisable to have attempted more at the first operation.

The ligations during the first operation were of the external and internal jugular, subclavian and innominate veins and the subclavian artery; and during the second operation of the axillary and long thoracic veins and subclavian, axillary and long thoracic arteries, a total of ten ligations (fig. 11).

Postoperative Notes.—On Aug. 11, 1928, the first dressing was applied.

On August 11, 3 a. m.: The patient felt well. There was no pain in the right hand or arm, which seemed even warmer than the other. The dressings which were loose were removed; a slight serosanguineous discharge was noted. The tissues were quite boggy in the region of the wound, suggesting edema. During

the operation it was noted that there were frequent little bursts of clear fluid into the wounds, apparently from dilated lymph vessels. The patient complained of being hot. Examination revealed a blood pressure of 170 systolic and 110 diastolic. A dry dressing was applied.

August 11, 10 a. m.: The wound was dressed. There was boggy swelling, suggesting accumulation of fluid in the wound. A probe was introduced into the wound and a considerable amount of almost clear fluid was evacuated. A dry dressing was applied. The blood pressure was 136 systolic and 84 diastolic. It was evident that the peak of the postoperative rise in blood pressure would have been missed if a reading had not been made during the night (3 a. m.). The pulse in the right radial artery was easily palpable for the first time since the patient was first seen; the rate was 84.

August 12: The dressing had to be changed on two occasions because of the marked serous discharge from the wound. No bleeding occurred. A few stitches were removed. The blood pressure was 164 systolic and 86 diastolic; the pulse was 86.

August 13: The remaining stitches were removed. The wound was in good condition, and no discharge had occurred since the day before. Apparently, there

Fig. 12 (November, 1928).—Complete cure, three months after the second operation. The circumferential measurements of both arms is the same. The patient writes, "I am feeling perfectly well."

was no further filing up of the wound. The blood pressure was 166 systolic and 84 diastolic; the pulse rate was 88. A dressing was applied.

From this time on, the progress of the patient was uneventful, with the exception of the development of an extensive thrombophlebitis of the veins of the anterior wall of the chest and of the upper arm. The condition subsided with rest in bed, immobilization of the arm and hot fomentations.

September 12: The patient left for Mexico. No thrill could be felt nor bruit heard. The shoulder and arm were steadily decreasing in size. The patient was feeling stronger daily. The blood pressure was 130 systolic and 86 diastolic. The pulse rate was 76.

November 11: The patient wrote: "I am feeling perfectly well and my arm is recovering its life. It is a little stiff but its size is normal at this time, as you will notice from the enclosed picture" (fig. 12).

COMMENT

Thompson, in his classic monograph on subclavian aneurysm, said that "ligature of the first part of the subclavian artery has turned out to be a very unsatisfactory operation. It is both difficult and dangerous.

The artery is very short, numerous branches arise from it, it is very deeply situated and is surrounded by important veins and nerves. The internal jugular and vertebral veins are directly in front of it; also the pneumogastric and phrenic nerves. At its origin, the right innominate vein forms an anterior relationship." I believe that the reason this procedure has proved so difficult is that the surgeon has attempted this ligation without first performing a subperiosteal resection of the inner half of the clavicle. By this step, a very difficult approach is rendered much more accessible.

In an arteriovenous fistula the pulse is smaller on the injured side showing that the volume of arterial blood is less, while the dilated veins show that the venous blood is increased.

The retardation of the pulse rate on closure of the fistula by digital pressure or by permanent suture or ligation was explained by Holman as follows: "Closure of the fistula results in a filling up of the aorta by the volume of blood which formerly leaked through into the veins.

TABLE 2.—*Circumferential Measurements During Second Stage of Treatment, 1928*

Area	Jan. 2	Feb. 28	Aug. 6	Nov. 12
	Cm.	Cm.	Cm.	Cm.
Wrist	17.0	17.0	17.0	16.5
Middle forearm, 14 cm. above the wrist	28.0	28.0	27.0	24.0
Elbow	32.0	32.0	32.0	28.0
Middle arm just below greatest swelling	38.0	38.0	37.5	31.5
Over greatest swelling upper third arm	41.0	41.0	41.0	35.5
Axilla	45.0	45.0	45.0	42.5
Right side of chest at level of midline	52.5	52.5	54.0	54.0
Left side of chest at level of midline	51.0	51.0	52.5	52.0

This produces an increase in aortic pressure which in turn stimulates the depressor fibers of the vagus in the arch of the aorta and reflex slowing of the heart is the result."

Holman and Dock have definitely shown, in experimenting with animals, that in an arteriovenous fistula the cardiac output is doubled. This observation is interesting as it differs from that of Lewis who stated "that the output in an arteriovenous fistula does not appear to be unusually altered."

Holman was among the first to emphasize the physiologic basis for the alteration in blood pressure by opening and closing the fistula, giving as his explanation an increase in the total volume of blood during the existence of the fistula.

Conditions in the fistula that determine or influence the systemic effects are, according to Matas: (*a*) the size of the arteriovenous fistula; (*b*) the volume and force of the arterial stream that is short-circuited into the communicating vein; (*c*) the caliber of the vessels involved; (*d*) the proximity to the heart; (*e*) the duration of the fistula; (*f*) the age of the patient, and (*g*) the coexistence of the antecedent cardiovascular disease.

It is always to be remembered that gangrene occurs much more frequently in an arteriovenous than in a saccular aneurysm. I believe that in an arteriovenous aneurysm simultaneous ligation of the accompanying vein with the artery should always be executed. This would appear at first thought to be paradoxic. Indeed, it reverses a surgical principle long emphasized by writers of textbooks. The special needle or ligature carrier was invented with the main idea of being able to ligate the artery safely without injury to the satellite vein. To ligate unhesitatingly a normal and large vein is contrary to tradition. Tradition is not easy to set aside.

I believe, however, that there is now sufficient experimental and clinical proof that this procedure today should be a recognized rule. Professor Tuffier, the great French surgeon, said that "ligation of the correspondingly healthy vein" should be executed 'in all cases of ligation of the great vessels of the root of the limbs." Sir George Makins, the eminent British surgeon, long an authority in surgery of blood vessels, stated that "the elimination, in fact, of the capacious main vein is a real advantage, since this for the time affords a too ready channel of exit for the diminished arterial supply, as well as an undesirable reservoir for stagnation." He concluded, "these considerations have led me not only to regard obligatory simultaneous occlusion of a main artery and vein as a negligible factor in the risk of gangrene of a limb; but to hold further, that the procedure is preferable whether the vein be wounded or not; the result of the combined procedure being to maintain within the limb for a longer period the smaller amount of blood supplied by the collateral arterial circulation, and hence to improve the conditions necessary for the preservation of the vitality of the limb." In accord with this procedure but disagreeing with Makin's explanation, Brooks and Martin stated that the increase in the peripheral blood pressure is the principal beneficial result of ligating the vein. Reid and Holman's observations support the latter view. Holman, in his brilliant experimental work, repeatedly proved the necessity of this simultaneous ligation of vein and artery.

In the light of this knowledge one must accept the view that some degree of equilibrium of the arterial and venous systems must be preserved. I believe that the following maxims may be laid down for the prevention of gangrene: 1. In an arteriovenous fistula or aneurysm it is imperative to ligate the accompanying vein if the artery is ligated. 2. In a simple aneurysm it is at least advisable to do so.

Few or none of these patients should be operated on until from three to six months have expired unless the cardiovascular effects are conspicuous and progressive. Then if the fistula still exists, further delay is not indicated. The reasons for waiting are (*a*) the possibility of a spontaneous closure of the artificial communication with cure, and

(*b*) the possibility of awakening a lurking infection if the operation follows the injury too soon. Infection usually results in death.

"Common errors in the treatment of aneurysms," according to Halsted, "are the following: (1) opening the sac or pulsating hematoma without first making a temporary occulsion of all the possible sources of hemorrhage; (2) the permanent ligation of a great arterial trunk as a precautionary measure in the search for a distal bleeding point; (3) ligation of a trunk too far from the aneurysm; (4) stuffing the wound with gauze to arrest hemorrhage; (5) drainage; (6) the employment of catgut for the ligature, or of silk that is too fine, and (7) ligation of the artery proximal to an arteriovenous aneurysm or fistula."

Surgeons attempting to intervene in aneurysms or arteriovenous fistulas should be much more concerned with hemostasis and meticulous technic than with the duration of the operation. These operations are at best always tedious and time-robbing and demand the best of the surgeon's skill. Without profuse hemorrhage these patients stand operations of six or seven hours' duration without severe shock. "An operator searching for a bleeding point in a pool of blood, and particularly so when embarrassed in his movements by the adherent walls of an aneurysm within which he is working so disadvantageously, presents a distressing spectacle. I would rather devote an additional hour or more to an operation than be caught for a few moments in such a predicament" (Halsted). The reestablishment of a normal vascular equilibrium is the objective sought.

SUMMARY

A case of traumatic subclavian arteriovenous aneurysm of eight years' duration showed:

1. Enormous swelling of the right side of the chest, the right portion of the shoulder girdle and the entire right arm and hand, with great distention of veins in these parts.

2. Cardiac dilatation and hypertrophy resulting from increased volume flow through the heart incident to the production of the fistula.

3. Branham's bradycardiac reaction and associated variations in blood pressure due to an increase in blood volume caused by the fistula.

4. Characteristic bruit and thrill transmitted centrifugally.

5. Increase of oxygen content in veins distal to lesion.

6. Dilation of the proximal artery, diminution of the distal artery and great dilatation and thickening of the distal vein.

7. That proximal ligations of artery and veins improved the condition but did not effect a cure.

8. That subsequent distal ligations, ten months later, completing the quadruple ligation, effected a cure.

ABSTRACT OF DISCUSSION

Dr. Hermann B. Gessner, New Orleans: Dr. Gilcreest is to be congratulated, not only on the skill with which he treated this patient, but on the thoroughness with which he reviewed the symptomatology and pathology of the condition. In my experience with arteriovenous aneurysms, which has been limited to those of the lower extremity, I have been able in some cases to control the fistula by attacking it from the venous side. I remember one interesting case in which the vein involved was a vena comes. I was able to incise the vein, suture the orifice with paraffinized silk, and then sacrifice the vena comes.

I think one diagnostic sign is worth bringing out. Dr. Gilcreest did not mention it, probably because the diagnosis in his case was so clear. I refer to the sign I have learned from my teacher and friend, Dr. Rudolph Matas: It is the change in the distal pulse, the Delbet sign. I can recall two cases in which the diagnosis was made in that way. One was a case of aneurysm of the popliteal artery. No bruit was discernible. A mass was present. It was not possible to find the pulsation of the tibial arteries. I presented this patient to a class and Dr. Matas operated, confirming the diagnosis. The explanation was simple. The aneurysm was so filled with clot that there was little movement of blood.

In a case of aneurysm of the forearm seen recently, the patient had a hematoma that permitted the diagnosis by means of a needle. There was no pulsation and no bruit; yet, on incising the hematoma, arterial blood came out, escaping from a wound of the radial artery. I think it important to realize that aneurysms may be recognized on this Delbet sign.

It happens not infrequently that an aneurysm is incised under the diagnosis of abscess. I know of a case in which an aneurysm of the subclavian artery was incised in that way. The patient had pain and tenderness, a rise in temperature and leukocytosis. A colleague of mine opened the aneurysm in the belief that it was an abscess. I think this diagnostic point should be brought out and carefully considered, as tending to prevent such errors.

Dr. Albert O. Singleton, Galveston, Texas: Through his persistence in this case Dr. Gilcreest has obtained an excellent result. Arteriovenous aneurysms are more of a problem than the sacculated aneurysms. Probably in Dr. Gilcreest's case he performed part of the operation and hoped for success, and then did the other part subsequently, rather than completing it all at once. He has shown that the operation took a long time, and I do not think that he is necessarily a slow operator. Any one who has operated in these cases realizes that although time passes rapidly, the surgeon often does not get far.

I appreciate Dr. Gessner's remarks about the diagnosis, and think they should be repeated often. In the usual run of work, these cases are not seen frequently, and these points are sometimes forgotten. The aneurysm varix has an opening, which makes the problem more difficult than in the case in which Dr. Gessner was able to suture the aneurysm from within the vein. I have been helped in two or three cases in making a differential diagnosis between aneurysm varix and arteriovenous aneurysm by injecting sodium iodide into the artery, and then making a roentgenogram to show the direct communication. This method also shows the collateral circulation in its approximation to the sac. In one case it showed that there was a considerable interval and a sac between the artery and the vein.

Dr. Gilcreest, I think, is right in ligating the vein at the same time as the artery. I had a patient with an aneurysm at the lowest part of the external iliac artery, on which I intended to perform a double ligation. On exposing it, however, above the aneurysm and clamping the artery I saw such an immediate blanching of

the foot that it was evident that an ischemic gangrene would probably develop if I ligated the artery alone. On clamping the vein, the warmth of the foot promptly returned. I ended by ligating both of the vessels, and the circulation was not much interfered with. The leg remained warm and continued warm until a subsequent ligation resulted in a perfect cure.

Dr. W. Lowndes Peple, Richmond, Va.: I also wish to congratulate Dr. Gilcreest on his perseverance and success, and wish to show you a recent case of aneurysm of the femoral artery, which occurred in a man about 22 years old, who had been shot with a 22 caliber rifle about ten years before. He was not ill at the time, there was no swelling, he resumed all of his activities, athletics and so forth, and had no trouble for nine years. He then began to have attacks of pain in the region of his heart. Six weeks before his admission to the hospital he had violent pain over the region of the iliac artery. All of the typical signs and symptoms of a fistula were obtained. The x-ray picture showed an enormously large artery pulled to one side, the femoral vein accompanying it also pulled to one side, and the vein which lay beneath it was visible. One could see the small aneurysm, about the size of a pigeon's egg. It was as though the drift of the arterial blood driven across the vein has caused this sacculation. The man was a good patient, young, strong and thin; it was possible, therefore, to ligate the artery above and below, and ligate the normal femoral vein above and below. We then dissected out the sac. There were several veins opening into this which were easily tied.

I am glad to see that the other speakers have no doubts whatever about the advisability of ligating even the normal accompanying vein. This seems to be now an ordinary procedure, which one might speak of as stabilization of the vein. There was no reaction in the dorsalis pedis artery or the posterior tibia which was apparently normal on both sides. The patient made an uneventful recovery.

Dr. J. L. Campbell, Atlanta, Ga.: I suppose that the surgical division of the Grady Hospital is the best place in the world to find injuries to blood vessels. I recall twenty-five or thirty patients who have been operated on for this condition in my service, and most of these cases were of the arteriovenous type. I operated on a patient for subclavian aneurysm a number of years ago, and am glad to know that Dr. Gilcreest recommends resection of the clavicle. I do not think that one can operate adequately without this space. In my case I succeeded in curing the patient, in whom the condition was quite recent, by proximal ligation. The patient was a negro and I was unable to keep track of him.

The next case was that of an aneurysm of the common carotid artery in which an arteriovenous aneurysm had occurred. In that case I succeeded in restoring both vessels and the patient recovered.

The next case was that of a subclavian aneurysm in which I attempted to restore the lumen of both vessels. I had just the condition that Dr. Peple showed, with dilation of the vein. I applied a suture and attempted to make the separation so that I could close it. The patient became weak and the anesthetist said that I had better stop. Unfortunately, the patient died. That is the only patient that I have lost.

I have had two cases of aneurysm of the profunda and two of the femoral artery. In one of these I attempted to restore the lumen of the vessel, and gangrene resulted. In the other case I did not see the patient until after the limb had been amputated. I have had one aneurysm at the bend of the elbow, and two or three in the popliteal space. When the patients are seen early I think proximodistal ligation should be performed. If they are seen later, but when the vessels can be cleared and the patient is in good condition, I think that the

lumen can be restored without much difficulty. In one case of a pulsating exophthalmos I succeeded in partially occluding the internal carotid artery with a band, and relieved the patient of all his symptoms.

These cases are interesting. Any time the physicians present happen to be in Atlanta, I shall be glad to show them some of the cases. There nearly always are several in the hospital.

Dr. George Tully Vaughan, Washington, D. C.: I listened with great pleasure to Dr. Gilcreest's account, for I have had much experience with aneurysms of various kinds. Dr. Campbell just said that he was foolish enough to try to close the wounds and vessels in one patient and the patient died. Perhaps if he had closed the wounds the patient would not have died. I was foolish enough to try the same thing a number of years ago. A patient had just been shot through Scarpa's triangle. The injury happened on a street car and someone stuck a handkerchief into the wound. I watched the case and for two weeks there was no sign. Then an aneurysm varix developed, with a bruit. I operated and found a wound in the vein and artery, also four points in vessels filled with lymph. I closed these openings with fine vaselized sutures. The patient is living today, after fourteen years, and has no trouble with his extremities.

So many surgeons have spoken of the necessity of ligating both vessels, that they occlude the veins. This may be necessary occasionally, but not always. I have operated on many patients and have performed the Matas operation on several occasions, filling in the walls of the vessel sac so as to close the vessel completely, and not touching the vein. I have done this on the external iliac, the iliac, and on the femoral arteries and have not seen a case of gangrene. I have had probably a dozen cases of this kind.

I wish to ask Dr. Gilcreest why he did not find the openings in these two vessels and sew them up instead of using so many ligatures.

Dr. Walter C. G. Kirchner, St. Louis: I wish to congratulate the essayist on this splendid piece of surgery. Three years ago I ligated a subclavian artery in the first portion, I wish to mention a few points in differential diagnosis. This case was also a gunshot wound, the aneurysm having enlarged and become engorged on the vessels of the neck. It was present on the left side. There was no pulsation in the right pulse. There was atrophy on the one side, in addition to the enlargement on the other. In other respects there were all the characteristic signs of aneurysm. Careful dissection was made under general anesthesia. It was possible to ligate the first portion of the subclavian artery near the aortic arch without resecting the clavicle. In fact, that was the easiest part of the operation. The more difficult part, a fact that I can appreciate greatly, is the opening of the sac and the treatment of the condition when the hemorrhage is present. Unless one has had that experience he cannot realize how difficult it is to find these structures. In the aneurysm there is a lot of fibrous tissue and a lot of disarrangement of structures. This explains why so much time is necessary in handling these cases.

Dr. Gilcreest: I am naturally pleased that the report of this case excited such extensive discussion.

Referring to Dr. Gessner's mention of Delbet's sign, it will interest him to know that the patient's radial pulse was practically imperceptible, which is consistent with what Dr. Gessner pointed out; it became palpable only after the second operation.

I am glad to note that Dr. Singleton also appreciated that to carry out this technic correctly consumes much time. The cases reported by him and Dr. Peple are of great clinical significance.

The large series of operations by Dr. Campbell are most unusual. I do not know anywhere in the country where such a wealth of records of vascular surgical procedures could be found. I shall indeed avail myself of Dr. Campbell's invitation to see some of these patients the first time I revisit Atlanta.

Answering Dr. Vaughan's question of why I did not find the openings into the arteries and veins and sew them up, instead of resorting to so many ligatures, I can only repeat what I said before, that to have attempted this procedure through such a wilderness of large dilated thin-walled veins would have been only to produce profuse hemorrhage and court disaster. Dr. Kirchner emphasized this in his remarks.

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THE CELIAC PLEXUS AND ITS BRANCHES*

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Modern surgery through the use of local anesthesia has focused attention on the celiac plexus. At first, the chief concern was merely to develop a technic for making injections into this plexus which would permit the carrying out of certain major operations under local anesthesia. This led to the recording of new observations concerning its physiology. Relatively little attention has been paid, however, to the exact macroscopic and microscopic appearance of this region in the light of recent surgical experiences. Some of the disturbances referable to the duodenum and the pancreas which make their appearance after operations on the gallbladder and other abdominal operations require an exact anatomic knowledge of the celiac plexus for their proper understanding. So, too, attacks of abdominal pain simulating pain due to gallstones can be better differentiated from pancreatic, celiac and other types of medical and surgical pain, if one appreciates more fully the histologic character and ramifications of this plexus.

The modern surgeon still refers to the anatomic plates of the celiac plexus executed during the past century by Hirschfeld-Leveillé,[1] Henle,[2] Testut,[3] Poirier,[4] Quain[5] and others, since nowhere in the newer literature is there to be found any record of a recent systematic investigation of the plexus as a whole. Even the work of Hirt[6] (1924) is concerned only with the innervation of the kidney and the suprarenal gland, while Raigorodsky[7] (1928) described only the branches of the

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hepatic plexus. The well known illustrations which are to be found in textbooks give, in general, a clear idea of the topography of the branches of the plexus. But they are more or less schematic, do not concern the innervation of the pancreas and the duodenum and do not treat of the plexus in relation to surgical procedures.

This stimulated us to investigate the celiac plexus from the standpoint of modern surgery, which we did with the help of the newest macrotechnical methods. We carried out our investigations on thin adult human cadavers. In animals, the topography and distribution of the plexus are extremely variable, so that the anatomic relationships, as well as the physiologic results, obtained from dissections and investigations carried out on animals cannot be compared with those obtained in man. It has been our aim in this study to present a systematic description of the plexuses and their connections without exact reference to topographic details.

TECHNIC

We dissected under slowly dripping water, and we also employed the acidol pepsin digestion method with good results.[8] The dissection was also carried out on specimens that were not fixed and which were kept in cold running water during a period often of from two to three days or more. This loosened the collagen fibers. The cleanly prepared nerves were later colored by the method described by Gyermek[9] of this institute in 1919.

In the preparation of fine nerves there is always the danger of calling adjacent lymph vessels, small veins and connective tissue fibers, nerves. To avoid this, we compared several specimens prepared one after another, and controlled the relation of the nerves, as well as of the ganglions, with one another.

In addition to the celiac ganglions, well known treatises also describe the celiac plexus, which they consider the central part of the whole system. The following are mentioned as individual parts of the plexus which go to the various organs: (1) plexus phrenicus or plexus diaphragmaticus, (2) plexus aorticus, (3) plexus suprarenalis, (4) plexus renalis, (5) plexus spermaticus, (6) plexus gastricus superior and inferior, (7) plexus hepaticus, (8) plexus lienalis, (9) plexus mesentericus superior, (10) plexus mesentericus inferior and (11) plexus pancreaticus. We, for the most part, employ this grouping.

In our description, we will consider: (1) the celiac ganglions, (2) the roots of the celiac ganglions, (3) the branches of the celiac ganglions and (4) the microscopic structure of the celiac plexus.

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8. Two pastils of strong acidol pepsin are dissolved in 200 cc. of water. The field of dissection is kept well moistened with this solution. It is applied either by tampons or by pipet and, if necessary, over a period of from two to three hours. This makes the removal of connective tissues considerably easier.

9. Gyermek: Farben makroskopisch-anatomischer Präparate, Ztschr. f. wissensch. Mikr. **35**:45, 1918.

THE CELIAC GANGLIONS

In most instances, we found a flat ganglion the size of a strawberry on each side of the celiac artery (figs. 1, 2 and 3), the form of which may be variable. We sometimes found two, three or more smaller ganglions on each side (fig. 4). The ganglions on one side are united to those on the other by many transverse fibers above and below the celiac artery, in front of the aorta (fig. 1). In the majority of cases, however, an unpaired ganglion lies under the celiac ganglions directly on the aorta (fig. 3 *18*). Many authors call this ganglion the ganglion aorticum or, because of its union with the renal plexus, the ganglion aorticorenale. The other ganglions in this region, which we have illustrated in our figures, belong to the diaphragmatic and renal plexuses. The celiac ganglions form the central point for the celiac plexus.

THE ROOTS OF THE CELIAC PLEXUS

The roots of the celiac plexus are formed by the nervi splanchnici majores and minores and both vagi. Figures 1 and 2 and figure 3 *2* and *2a* show clearly the course of the splanchnic nerves. It is of practical importance to note the lengthy course (from 8 to 10 cm.) of the nervus splanchnicus major (fig. 3 *2*) behind the lumbar portion of the diaphragm. Surgical procedures also make use of this fact. Thus, the splanchnic nerves are infiltrated transdiaphragmatically in the space between the vertebral column and the lumbar portion of the diaphragm. In that the splanchnic minor (fig. 3 *2a*) lies close to the splanchnic major, it is of course blocked by the same injection. In this way (as will be seen in the paragraph dealing with the microscopic structure of the plexus), all the sensory roots of the celiac plexus are blocked. Figure 3 *3* shows that after the splanchnic major perforates the diaphragm, it then turns in a right angle medially and ends in the celiac ganglion.

One reads in most textbooks (for example, that of Spalteholz [10]) that the left vagus does not always take part in the formation of the celiac plexus. We, however, found in every specimen that both vagi contributed many branches to the formation of the plexus (fig. 1 *3*).

Spalteholz pointed out that the upper lumbar ganglions of the sympathetic send fibers to the plexus. Because of the proximity of the plexus to the ganglions, this would seem quite possible. We found no such branches.

THE BRANCHES OF THE CELIAC PLEXUS

We desire to draw special attention to the linking branches between the celiac ganglions of both sides (fig. 1) which lie in front of the

10. Spalteholz: Handatlas der Anatomie des Menschen, Leipzig, S. Hirzel, 1895-1903.

aorta, surround the celiac artery and form a dense plexus. The older descriptions lead one to the erroneous belief that both celiac ganglions sometimes form a single semilunar ganglion. We did not observe this in any specimen. As we have already pointed out, the celiac ganglions form with their branches the center and chief plexus of nerves going to the abdominal organs. The splanchnic nerves enter the ganglions, while the fibers of both vagi either end in these linking branches or run direct to the plexuses in the organs. All sympathetic and sensory fibers of the celiac plexus arise either from the ganglion bodies or from the linking branches. This explains why infiltration of the celiac ganglions results in complete anesthesia of the area between the diaphragm and the pelvis.

We refer briefly to the following plexuses of nerves going to the various abdominal organs.

Plexus Phrenicus (figs. 1 and 2).—This small plexus lies on the anterior part of the lumbar portion of the diaphragm in the region of the inferior phrenic artery. The roots of this plexus arise directly from the celiac ganglions. We found the ganglion phrenicum, which is the size of a small pea, on both sides (fig. 1 *12*). On the left side, the ganglion phrenicum lies in closer relation to the celiac ganglion. Luschka,[11] in 1853, described the relation of this ganglion with the phrenic plexus and phrenic nerve. We observed the same relationship in every specimen. But the phrenic plexus and the sympathetic nervous system play no part in the motor innervation of the diaphragm. Our microscopic investigations have shown that the branches of this plexus consist mostly of nonmedullated sympathetic fibers, and that between the sympathetic fibers only solitary medullated fibers are to be seen. The sympathetic fibers run to blood vessels and to the suprarenal glands, while the medullated fibers unite the region of the celiac plexus with the phrenic nerve and through the phrenic nerve with the cervical plexus. This is the route through which "pain" from the region of the celiac plexus (liver, pancreas, kidney, stomach and intestinal canal) reaches the plexus in the cervical region and produces reflex pain in the shoulder. In support of our observation is also the fact that, after splanchnic anesthesia, the movement of the diaphragm is in no way affected either by the development of a partial paralysis or by other functional disturbances.[12]

Plexus Suprarenalis (figs. 1 *13* and 2 *9a*).—The many roots of this plexus arise from the celiac ganglion and from the phrenic and renal plexuses. We found no macroscopic ganglions in this plexus. Accord-

11. Luschka: Nervus Phrenicus, Tübingen, H. Laupp, 1853.

12. Kiss and Ballon: Contribution to the Nerve Supply of the Diaphragm, Anat. Rec. **41**:285, 1929.

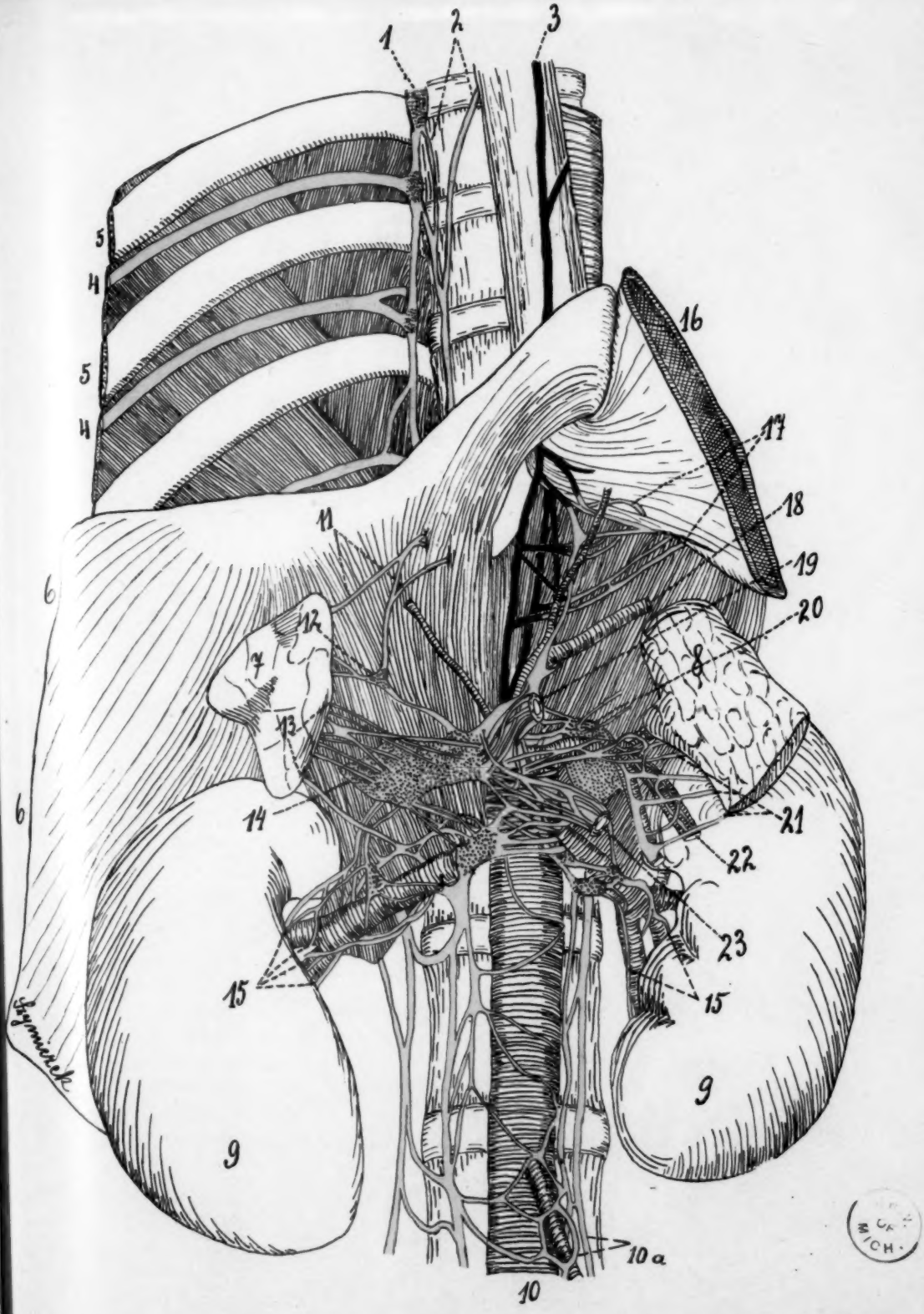

Fig. 1.—Plexus coeliacus (splanchnic—deep level). Figures 1 and 2 are taken at different levels. *1* indicates truncus sympathicus; *2*, nervus splanchnicus major; *3*, nervus vagus (right); *4*, nervi intercostales; *5*, ribs; *6*, diaphragm; *7*, suprarenal gland (right); *8*, pancreas (body), retracted to the left; *9*, kidney; *10*, plexus aorticus; *10a*, aorta; *11*, plexus phrenicus (diaphragmaticus); *12*, ganglion phrenicum; *13*, plexus suprarenalis; *14*, ganglion coeliacum (right); *15*, plexus renalis and renal artery; *16*, cardia ventriculi; *17*, plexus cardiacus; *18*, gastric artery (left); *19*, plexus coeliacus and coeliac artery; *20*, ganglion coeliacum (left); *21*, plexus pancreatis (left portion); *22*, plexus lienalis and splenic artery, and *23*, plexus mesentericus superior and superior mesenteric artery.

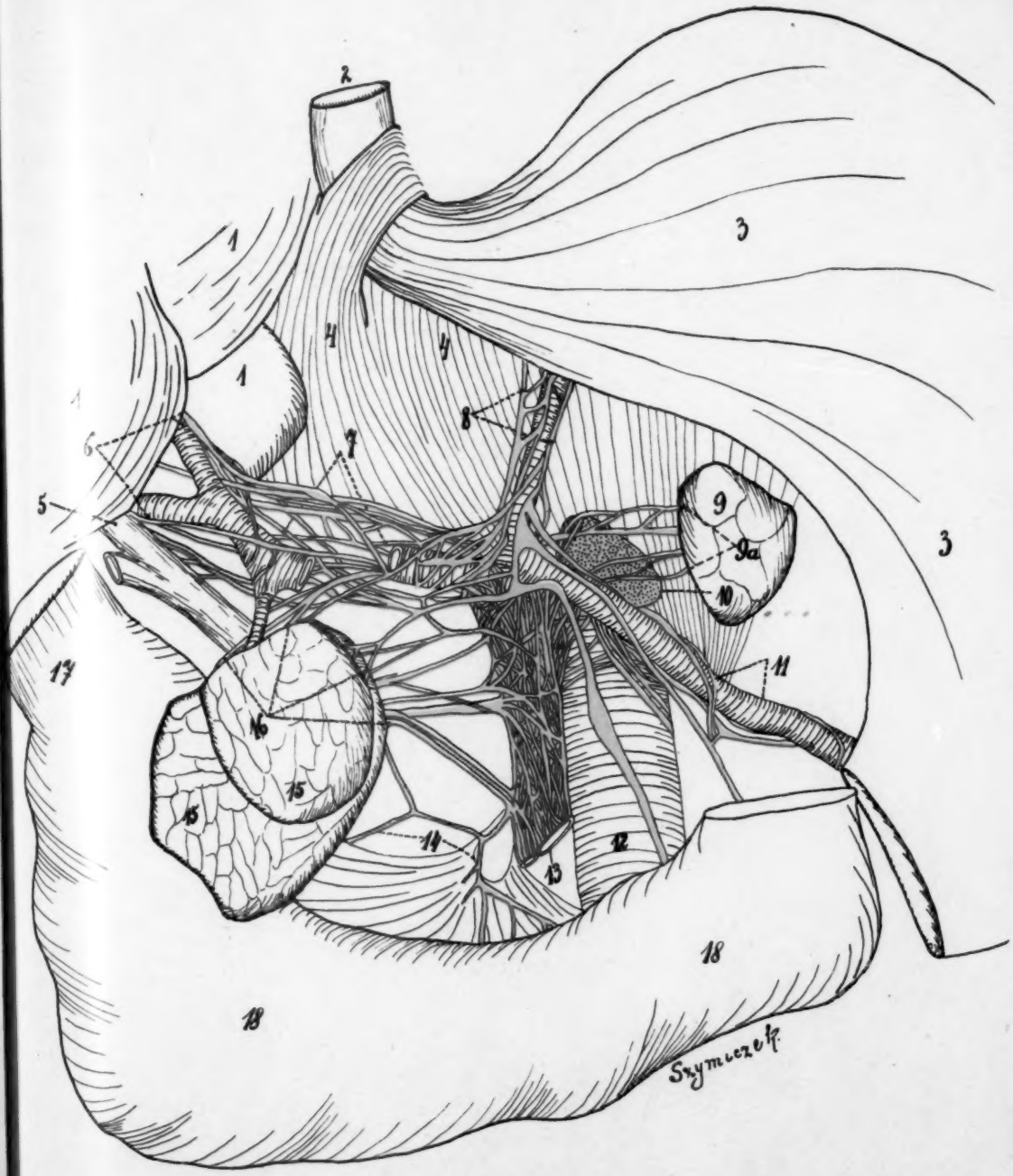

Fig. 2.—Plexus coeliacus (superficial level). *1* indicates liver, lobus caudatus (Spigelii); *2*, esophagus; *3*, stomach; *4*, diaphragm; *5*, ductus hepaticus; *6*, hepatic artery; *7*, plexus hepaticus; *8*, plexus cardiacus; *9*, suprarenal gland (left); *9a*, plexus suprarenalis sinister; *10*, ganglion coeliacum sinister; *11*, plexus lienalis and splenic artery; *12*, aorta; *13*, plexus mesentericus superior and superior mesenteric artery; *14*, plexus duodenalis (in part); *15*, caput pancreatis; *16*, plexus pancreatis (right portion); *17*, pylorus, and *18*, duodenum.

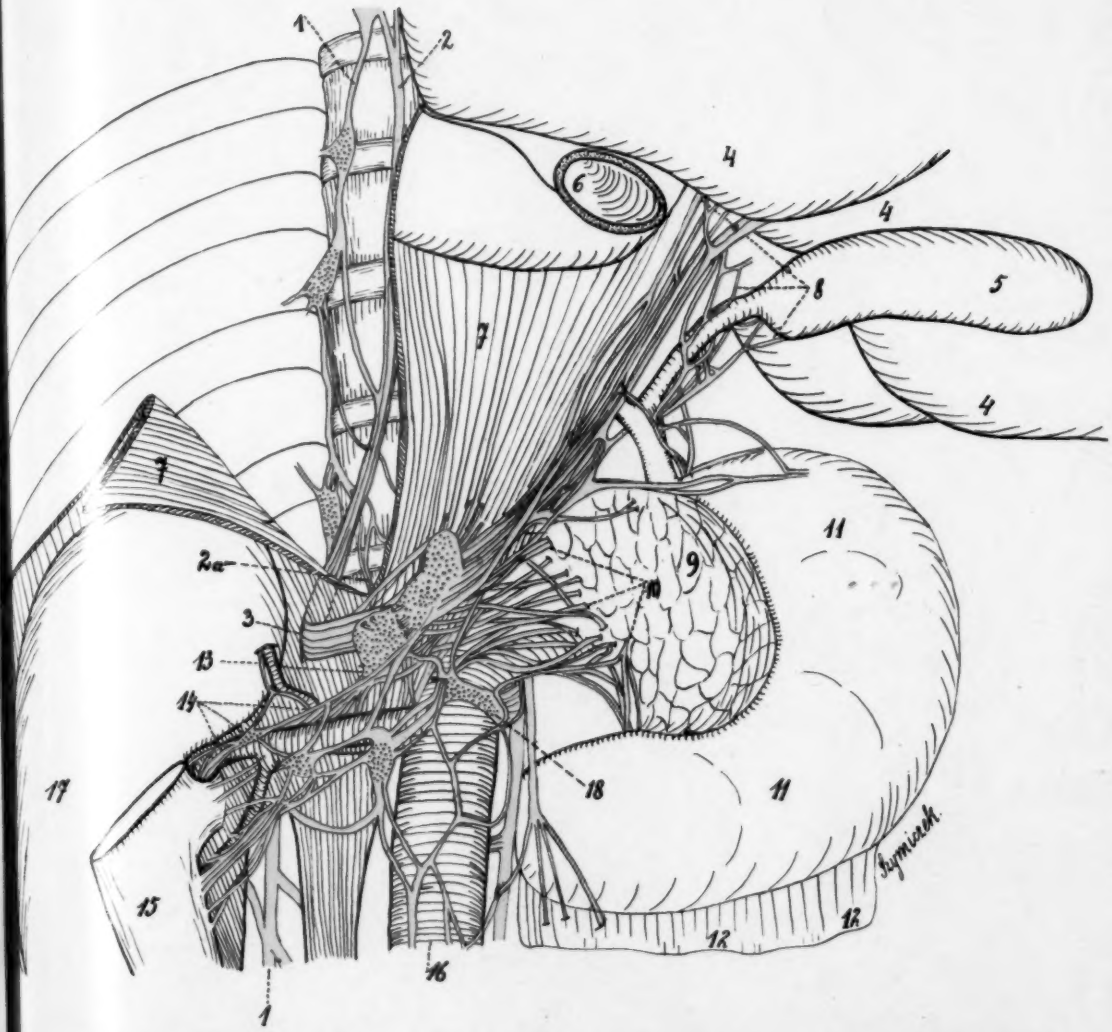

Fig. 3.—Plexus coeliacus, seen from the right. *1* indicates truncus sympathicus dexter; *2*, nervus splanchnicus major; *2a*, nervus splanchnicus minor; *3*, nervus splanchnicus major perforating the diaphragm; *4*, liver; *5*, gallbladder (pulled to left); *6*, vena cava inferior; *7*, diaphragm (lumbar portion); *8*, plexus hepaticus (posterior view); *9*, caput pancreatis (pulled over to left); *10*, plexus pancreatis (posterior view); *11*, duodenum (pulled to left, posterior view); *12*, peritoneal covering of duodenum; *13*, ganglion coeliacum dexter; *14*, plexus renalis dexter and renal artery; *15*, vena cava inferior (pulled to right, posterior view); *16*, aorta with plexus aorticus; *17*, kidney (right), and *18*, ganglion aorticum.

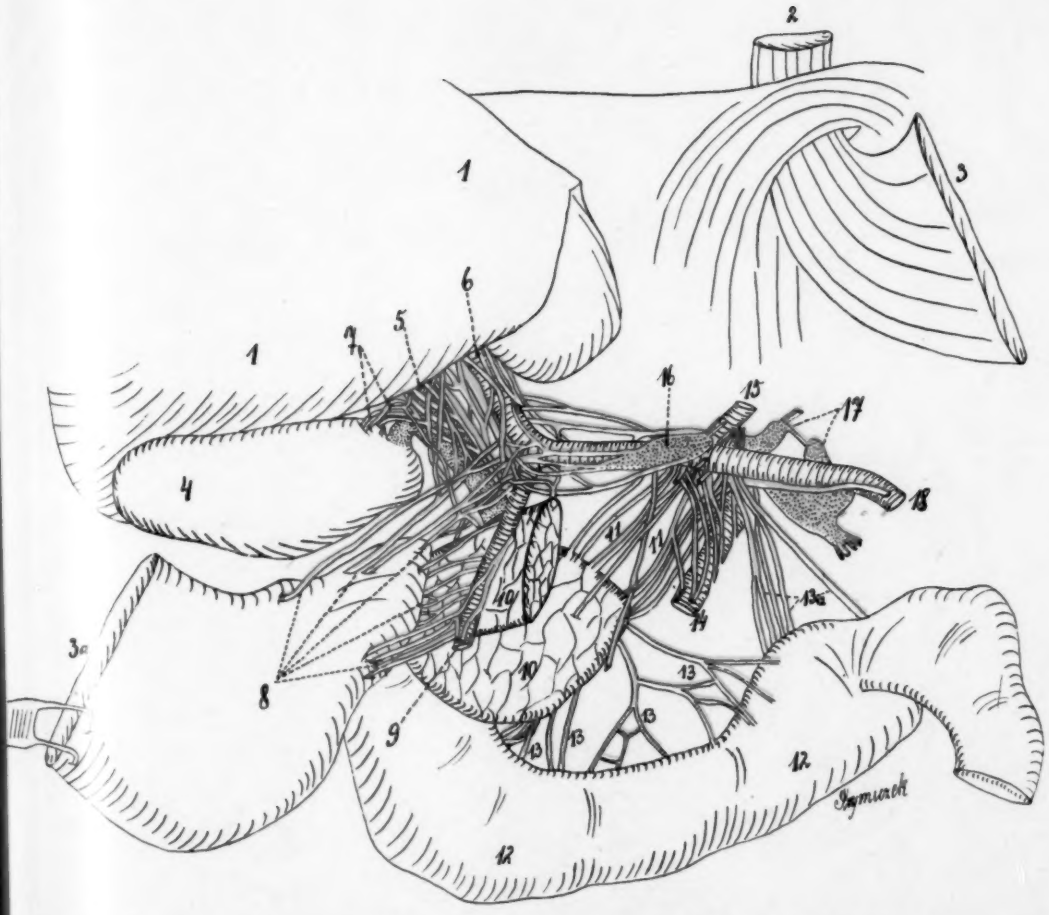

Fig. 4.—Plexus hepaticus, pancreaticus and duodenalis. *1* indicates liver; *2*, esophagus; *3*, stomach, cardia; *3a*, pylorus; *4*, gallbladder; *5*, ductus hepaticus and plexus hepaticus; *6*, hepatic artery and plexus hepaticus; *7*, plexus cysticus; *8*, plexus pyloricus; *9*, gastroduodenal artery; *10*, caput pancreatis; *11*, plexus pancreaticus; *12*, duodenum; *13*, plexus duodenalis; *13a*, plexus duodenalis (left portion); *14*, plexus mesentericus superior and superior mesenteric artery; *15*, gastric artery (left); *16*, ganglion coeliacum dexter; *17*, ganglion coeliacum sinister, and *18*, splenic artery.

ing to Hirt,[6] the individual fibers of the plexus run through the left suprarenal gland toward the renal cortex.

Plexus Renalis (figs. 1 *15* and 3 *14*).—The roots of this plexus arise from the celiac ganglion, from the uniting branches between the right and the left celiac ganglions and from the aortic plexus. We always found several ganglions in the plexus, varying in size from that of a millet seed to that of a pea. The branches follow the renal arteries.

Plexus Aorticus (figs. 1 *10* and 3 *16*).—This is a dense plexus on both sides of the abdominal aorta with many linking branches between both sides. Its chief branches arise from both trunks of the sympathetic, from the celiac plexus and from both renal plexuses. The aortic plexus sends branches to the inferior mesenteric plexus and to the spermatic plexus (latter not illustrated in the figures).

Plexus Cardiacus and Plexus Pyloricus (figs. 1 *17*, 2 *8*, 4 *8* and 5 *1*).—The descriptions hitherto make mention of a gastric plexus or a superior and an inferior gastric plexus. We constantly found that the nerves reach the stomach at the cardia and at the pylorus, so that it is preferable to speak of a cardiac and a pyloric plexus.

The cardiac plexus is formed by branches of both vagi and by branches from the celiac plexus. Accessory branches come from the celiac ganglions, from its uniting branches and from the hepatic plexus. It forms a small plexus, which encircles the gastric artery, and which runs to the cardiac portion of the stomach. Here it meets the branches of both vagi.

The pyloric plexus (fig. 4 *8*) comes from the hepatic plexus and from the plexus that winds around the gastroduodenal artery (fig. 4 *9*). The roots of the last mentioned plexus arise partly from the hepatic plexus and partly directly from the celiac plexus. The branches penetrate obliquely into the wall of the stomach.

We do not wish to make any lengthy clinical digressions in this report. But the description that has been given suggests that certain derangements after operations on the stomach may be explained on a purely anatomic basis, particularly the normal correlated action of pylorus and cardia. When one appreciates that gastrojejunal ulcer develops more frequently when a gastro-enterostomy has been performed on a normal stomach than when it has been performed for ulcer, and that amputation neuromas may be demonstrated at the base of gastric ulcers, then it seems not unlikely that at least some of the disturbances noted, particularly after extensive operations on the stomach, are to be attributed to the destruction of important nerve fibers. This may be caused either directly through degeneration of nerve fibers or indirectly by the formation of scars. Certain it is, however, that extensive resec-

tions of the stomach are not possible without destruction of important nerve fibers.

Plexus Lienalis (figs. 1 *22*, 2 *11* and 4 *18*).—The roots of this plexus come from the celiac plexus, from the cardiac plexus and from the superior mesenteric plexus. Single branches come directly from the left celiac ganglions. The splenic plexus directs itself with the splenic artery to the spleen. On its way, this plexus gives off many branches to the pancreas (fig. 1 *21*). We found no macroscopic ganglions in this plexus.

Hepatic Plexus (figs. 2 *7*, 3 *8*, 4 *5*, *6* and *7* and 5 *4*).—The hepatic plexus is a rich plexus that lies in the hepatoduodenal ligament, and surrounds the hepatic artery, portal vein, hepatic duct and common bile duct. As many fibers run anteriorly to these structures as posteriorly; for this reason Raigorodsky,[7] in a new description, distinguished an anterior and a posterior hepatic plexus. The fibers which go to the gallbladder form a small cystic plexus in the region of the cystic duct (fig. 4 *7*). The roots of the hepatic plexus arise from the right celiac ganglion and from the fibers which unite both celiac ganglions, from the aortic plexus and from the right renal plexus. The hepatic plexus sends branches to the liver, gallbladder, suprarenal glands, pyloric end of the stomach and head of the pancreas. We found no macroscopic ganglions in this plexus.

Raigorodsky differentiated four types of hepatic plexus after the four types of hepatic artery described by Rio-Branco.[13] One of us (F. K.[14]) recently described twenty-four variations of the hepatic artery, and we were able to compare our nerve specimens with his original specimens. We found that several branches of the hepatic plexus always follow the abnormal branches of the hepatic artery. The main part of the plexus, however, is situated even in such cases on the anterior and posterior surfaces of the common duct and the portal vein.

The purpose of the following is merely to point out that certain clinical applications can be made of our anatomic observations. We mention some of them:

Relatively little damage is done to the important nerves in operations confined to the cystic duct.

In operations on the common bile duct, it is possible to damage important nerve fibers. It is true that in conditions involving the common duct, the pain is often pancreatic in origin. This need not,

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13. Rio-Branco: Essay sur l'anatomie et la médecine opératoire du tronc coeliaque, Paris, 1912.

14. Kiss and Mihálik: Ueber die Zusammensetzung der peripherischen Nerven und den Zusammenhang zischen Morphologie und Funktion der peripherischen Nervenfasern, Ztschr. f. Anat. u. Entwgesch. **88**:112, 1928.

however, necessarily be so. If the same pain for which the patient was operated on persists, it is, of course, impossible to exclude with absolute certainty a pancreatic origin, even though the pancreas is clinically normal, particularly since the common bile duct is so closely related to the head of the pancreas. It is for this reason, too, that patients with conditions involving the common duct often complain of pain in the back. This pain can frequently not be differentiated from pancreatic pain or celiac neuralgia. That there should be pain in the back and vertebral tenderness is to be expected, since the gland, as well as important nerve fibers, lies retroperitoneally. Should, however, a new type of pain make its appearance after operation, one must always appreciate the possibility of some inflammatory or degenerative process involving the pain-bearing fibers which surround the common duct and should remember that this may be as frequent an occurrence as the ordinary scar formation in the common duct. The distribution of these fibers is such that an uncomplicated picture will only be present early in the cases, before there is an associated pancreatitis. Thus, in the more chronic cases, only a careful clinical history and tracing of the course and distribution of the pain will help to locate its original site.

Plexus Mesentericus Superior (figs. 1 *23*, 2 *13* and 4 *14*).—This plexus forms a shell around the superior mesenteric artery. All the fibers arise from the celiac plexus (ganglions and linking fibers). The vast majority of the fibers run with the superior mesenteric artery to the intestines. Individual fibers run to the hepatic, cardiac, renal, pancreatic and duodenal plexuses.

Plexus Pancreaticus (figs. 1 *21*, 2 *16*, 3 *10* and 4 *11*).—This rich plexus is not even mentioned in most textbooks and has never been illustrated. The plexus is made up of two parts. One part supplies the head of the pancreas, the other part the body of the pancreas. The part that supplies the head of the pancreas (figs. 2 *16*, 3 *10* and 4 *11*) arises with many trunks from the celiac plexus (ganglions and connecting fibers) and from the superior mesenteric, aortic, hepatic and duodenal plexuses. The main part of the plexus lies behind the head of the pancreas and sends fibers from there into the head of the pancreas.

The other portion of the plexus, which goes to the body of the pancreas (fig. 1 *21*), arises from the splenic plexus, and its fibers enter, one after the other, the substance of the pancreas. Both portions are without macroscopic ganglions.

This distribution of nerves to the pancreas, through its substance and thence to the concavity of the duodenum, explains clearly why acute pancreatitis often simulates ileus and why the latter may be an associated observation. We have seen that in certain cases these nerves may be involved by scar, which may spread between the nerve fibers or even produce amputation neuromas. We suggest the foregoing possibilities

merely as some of the causes of pain in chronic pancreatitis. The celiac plexus may itself be involved by some inflammatory process or through pressure. The former also serves to explain certain types of pain that persist after operations on the biliary system. Pain in pancreatic conditions, particularly cysts, may be due to a "neuralgia" of the pancreatic and celiac plexuses. It may, on the other hand, be due to direct pressure

Fig. 5.—Hepatic, cardiac and duodenal plexus, anterior view. *1* indicates cardiac plexus; *2*, caudate lobe of liver; *3*, liver; *4*, hepatic plexus; *5*, right branch of hepatic artery; *6*, gastroduodenal artery; *7* and *9*, duodenal plexus; *8*, duodenum; *10*, stomach; *11*, left gastric artery, and *12*, pancreas.

on nerve endings rather than to pressure and pull on the intestine. Of pain that is pancreatic in origin, it can usually be said that the localization corresponds to the organ from which it emanated. The retroperitoneal position of the pancreas and of important nerve fibers explains, as we

have already mentioned, why there is pain in the back and why pancreatic pain is often increased when the patient is in the dorsal position. It seems that conditions of the pancreas limited to the head of the pancreas cause pain more frequently than those situated elsewhere in the pancreas. The relationship of the common bile duct and the presence of a separate plexus going to the head of the pancreas may perhaps explain this observation.

Plexus Duodenalis (figs. 2 *14*, 4 *13* and *13a* and 5 *9*).—This plexus is not mentioned or illustrated in atlases or textbooks. The plexus lies behind the pancreas on the posterior abdominal wall in the retroperitoneal tissue. The fibers arise from the pancreatic plexus and from the superior mesenteric plexus; a separate portion from the celiac plexus goes to the duodenal jejunal flexure. The branches reach the duodenum on its concavity. The plexus is without macroscopic ganglions. The distribution and arrangement of nerve fibers going to the duodenum suggest a route by which duodenal conditions may be reflected in the pancreas.

Plexus Mesentericus Inferior (fig. 1 *10a*).—This plexus arises from the aortic plexus and surrounds the artery that bears its name.

Plexus Spermaticus and Ovaricus (not illustrated).—The fine trunks of this plexus arise from the aortic and renal plexuses. The plexuses run with the similarly named artery.

THE MICROSCOPIC STRUCTURE OF THE CELIAC PLEXUS

The trunks of the splanchnic nerves and vagi bring three different morphologic kinds of fibers to the celiac plexus. The nonmedullated sympathetic fibers preponderate in the splanchnic nerves (fig. 6 *2*). Among these are mixed many thin medullated fibers (fig. 6 *1*) and also solitary, thick medullated fibers (fig. 6 *4*). In the abdominal part of the vagus, we found the same three kinds of fibers (fig. *7*).

Kiss and Mihálik [14] (1928) showed that the sensory and parasympathetic (vagus) fibers have thin medullary sheaths, that the motor fibers have thick medullary sheaths and that the sympathetic fibers are nonmedullated. These authors also found fibers with thick medullary sheaths in the posterior spinal roots, the function of which is unknown. On the basis of these and the aforementioned investigations, the thin medullated fibers in the splanchnic nerve are known to be sensory, whereas the thin medullated fibers in the vagus are parasympathetic, and the large solitary medullated fibers of the splanchnic nerves probably belong to those leaving the spinal cord through the posterior roots and probably conduct themselves centripetally. The most important fact which the surgeon has to know is that the sensory fibers to the celiac plexus run in the splanchnic nerves; that is why so many thin medullated

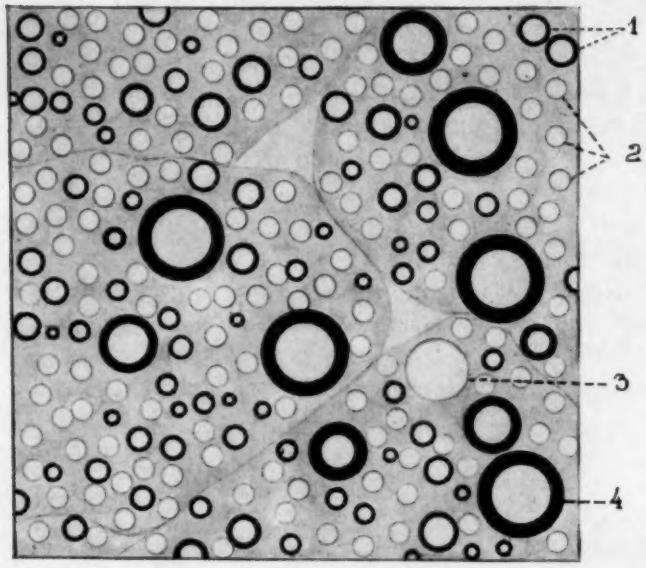

Fig. 6.—Transverse section of the nervus splanchnicus major sinister of an adult (osmic acid). *1* indicates thin medullated fibers; *2*, unmyelinated (sympathetic) fibers; *3*, capillary, and *4*, thick medullated fibers.

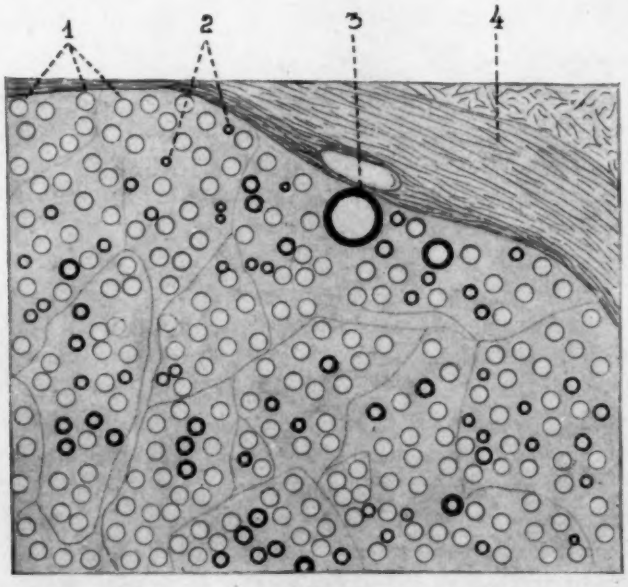

Fig. 7.—Transverse section of the abdominal portion of the left vagus nerve of an adult (osmic acid). *1* indicates unmyelinated (sympathetic) fibers; 2, thin medullated fibers; *3*, thick medullated fibers, and *4*, perineurium.

fibers are found in them. It follows from these observations that one finds in the branches of the celiac plexus the same three types of fibers that occur in the splanchnic nerves and in the vagi. In some plexuses, for example, the hepatic and renal plexuses, one finds fewer medullated fibers than one does in the branches of the superior and inferior mesenteric plexuses.

SUMMARY

A description of the celiac plexus from the standpoint of modern surgery is presented.

The celiac ganglions, the roots of the celiac ganglions, the branches of the celiac ganglions and the microscopic structure of the celiac plexus are discussed.

The plexuses of nerves going to the various abdominal organs are also mentioned. Particular attention is drawn to the descriptions and illustrations of the pancreatic and duodenal plexuses. These plexuses have never been illustrated before.

A block anesthesia of the splanchnic nerves gives the same results as infiltration of the celiac ganglions with their uniting branches.

In complete anesthesia of the splanchnic nerves or of the celiac plexus, the sensory, sympathetic and parasympathetic fibers are all blocked.

In order to obtain complete anesthesia, injections must be made on both sides of the splanchnic nerves, or in the celiac ganglions. A single injection suffices, because the splanchnic nerves, as well as the celiac ganglions, lie in loose retroperitoneal tissue.

We have drawn attention to the rich hepatic and pancreatic plexuses, which lie in the field of so many surgical procedures. Important fibers may thus be easily damaged.

It follows from the intimate relationship of the various plexuses with one another and with the centrally placed celiac plexus that certain lesions involving individual plexuses, such as the hepatic or the pancreatic plexus, may have their signs and symptoms reflected in other organs. Some disturbances referable to the duodenum and the pancreas after operations on the gallbladder may be explained on this basis.

THE BRANCHIAL APPARATUS

ITS EMBRYOLOGIC ORIGIN AND THE PATHOLOGIC CHANGES TO WHICH IT GIVES RISE, WITH PRESENTATION OF A FAMILIAL GROUP OF FISTULAS *

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Little has been written in this country concerning branchial cleft cysts and fistulas. Phases and fragments of the subject have been discussed in a few worthy papers. The interest aroused in a familial group of branchial fistulas recently studied in this hospital led us to feel that it would be worth while to present a fairly complete and comprehensive review of the whole subject in one paper.

Ninety case reports were studied, as well as the seventeen in our own archives. Many other case reports were not listed because of their incompleteness and because they presented nothing new.

Much of the information concerning the history of this subject was obtained from Wenglowski's extensive work.

HISTORY OF THE SUBJECT

The study of fistulas of the neck up to the presentation of von Ascherson in 1832 was in its infancy and in a chaotic state. After compiling his cases of lateral fistulas with all the known cases of fistulas and cysts at that time, von Ascherson concluded that these fistulas resulted from malformed branchial clefts which failed to close or to unite normally.

In view of the work of von Rathke, von Ascherson was cognizant of a prominent process that developed out of the second branchial arch in vertebrate embryos and that is the most prominent portion of the branchial apparatus; that is, the "Operculum branchiale of Rathke." Ascherson stated that the external orifices of fistulas pointed up under the operculum and thus the fistula was formed by a failure of the cleft boundaries to fuse or close. Succeeding authors, though they believed in the branchial cleft origin of the anomaly, opposed von Ascherson somewhat by introducing the conception that a fistula could develop from any of the four clefts. The fact that prompted this deduction was the

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variety of positions that the external orifice might and did take, being limited, as they knew, only by the region of the styloid process and sternoclavicular junction.

According to von Heusinger, the internal orifice of all fistulas was situated at the lateral wall of the pharynx at the base of the tongue. He also believed that the location of the external orifice determined which cleft was at fault, and stated that the fourth cleft was responsible in a case in which the orifice was at the sternoclavicular junction, because by dissection he had found that it coursed along the medial edge of the sternocleidomastoid muscle to the upper border of the larynx and then made a sharp turn toward the midline, opening at the root of the tongue.

Bland-Sutton and von Cussett devised schemas according to which one could determine from which of the four clefts a fistula developed. His doubted the truth and value of these schemas and maintained that the existence of a fistula pointing into the sinus praecervicalis but not into the pharynx is normal in the five weeks' embryo. Later the thymus develops from a portion of the sinus praecervicalis forming a connection between the two. If, he stated, the existing fistula does not grow downward in the thymus region but grows upward, then a true pathologic fistula exists. His, in 1889, changed his mind and stated that the thymus developed from the pharyngeal pouch and not from the precervical sinus.

Rabl refuted His, saying that the fourth cleft could not be a factor in the formation of fistula, because it is separated from the sinus praecervicalis by a thick mesodermal layer. The fistula could not result from the third because it is concerned with the formation of the thymus and therefore the fistula would have to be in close relation with the thymus, which is not true. He paid special attention to the origin of the viscera or organs of the neck from the second arch, since this arch is larger than the others. The second groove consists of a long irregular passage in which two rows of closely opposed epithelial cells divide the pharynx from the outside. He termed this the branchial canal and believed it to be the basis of the formation of fistula only, of course, when the epithelial membrane ruptures and therefore allows communication of the pharynx with the sinus praecervicalis.

In 1890, Kostaniecki and Milecki studied the literature in detail paying special attention to Rabl's work. They did not agree with the schemas of Heusinger, Bland-Sutton, etc., for the external opening of the fistula may at one time be high up near the jaw, then may close and open later in a lower position, and they therefore thought it foolish to say that the position of this opening had such significance as was claimed. The inner opening into the pharynx, however, is constant for any particular cleft or groove. The most frequent and constant internal opening is located in the supratonsillar fossa, and they concluded from a search of the literature that this corresponded with the second arch.

thus agreeing with Rabl. According to these two men, no branchial cleft could persist in toto, nor could in itself be the cause of a fistula. In the case of the complete fistula, the limiting membrane must be ruptured, and in this way the pharyngeal pouch must first break into the cervical sinus. Thus the complete fistula originates from a rupture of the second pharyngeal pouch into the cervical sinus.

Although later work was based on the observations of these authors, yet some persisted in believing that the first, third or fourth grooves were occasionally the origin of cysts and fistulas. Kostaniecki and Milecki believed that Virchow's case in 1865 originated from the first because of certain coexisting anomalies of the ear.

They, however, denied the validity of Watson's argument concerning a case in which the fistula followed the course of and ran in the stylopharyngeus muscles. That is, he concluded that since the stylopharyngeus muscle originates from the third arch, the fistula must have originated from the third cleft. The refutation was based on the fact that the musculature develops at too late a period to be of significance in this way.

Wenglowski held that if the lateral fistulas belong to the second groove, then the inner openings must be within the limits of this groove or cleft, that is, the arcus palatoglossus muscle (anterior border) and the arcus palatopharyngeus (posterior border). Never, he added, has an inner opening of a fistula been found within the tonsil or between the two limits mentioned. In the cases of Kostaniecki and Milecki, Koztowski, Kotalicki, Neuhöfer, Mobitz, Watson, Rehn, etc., the inner opening pointed at the root of the tongue (unterhalb) in the tonsil fossa and behind the arcus palatoglossus. The inner openings of the lateral fistulas, therefore, since the arcus palatoglossus is formed by a deeply embedded muscle belonging to the third arch, are closely related with the third and not the second arch.

Kostaniecki and Milecki and their advocates argued that the outer opening may be found anywhere and was of no import as regards the origin of the fistula—that the outer opening developed from the inflammation, suppuration and consequent rupture of the inflammatory mass, and the rupture might be at any height or position, depending on the depth or extension of the suppurating cyst or fistula.

Again, to the contrary, Wenglowski stated that after studying a series of cases, one becomes cognizant of the fact that the external openings point in a definite place as a rule. That is, they occur along a line extending from the angle of the mandible to the middle of the sternum, along the medial border of the sternocleidomastoid muscle. If suppuration and inflammation were factors in determining the orifice, it would be only logical to expect it to point anywhere and not follow a definite course as we know it does. That is, they could open lateral to the sternocleidomastoid muscle into it or even into the pharynx and esopha-

gus. In further opposition to Kostaniecki and Milecki, Wenglowski contended that the branchial apparatus does not spread itself into the viscera of the neck but belongs to the head. The second, third and fourth branchial arches do not extend downward but backward or posteriorly. It is self-evident that the branchial arches, as well as the grooves, are definitely and precisely fixed anatomically and topographically. If the formation of the fistula were due to the branchial clefts remaining open, then the fistula would have to open in the region of the submaxillary angle and there would be no basis for its opening lower in the neck. Then again some cases are found in which the external opening is located at the medial edge of the sternocleidomastoid muscle and in exploring the canal one finds that it does not lead upward but downward to the sternum, where it ends blindly. These cases, he contended, cannot have any connection with the branchial apparatus.

History with Special Reference to the Development of the Branchial Apparatus.—The first accurate embryologic description of the branchial apparatus in vertebrates was given by Rathke, in 1825. He described the arches and clefts in the three weeks' embryo of a pig and compared them with the branchial apparatus of the shark.

In 1827, von Baer described four clefts in the human embryo.

In 1832, von Ascherson for the first time discussed the origin of fistulas of the neck from the branchial apparatus, while in 1877, von Cusset made a compilation of various pathologic conditions of the neck independent of the branchial apparatus. He claimed that in man the first arch develops on the fifteenth day and the fourth during the first half of the second month. All arches extend to fuse with their fellows of the opposite side. Their substance consists of embryonic tissue lined externally with flat and internally with cylindrical epithelium. All arches arise in the region of the base of the skull, but because of the rapid growth of the first the lower four are crowded caudally. The rims or borders of all four grooves or furrows are definitely delineated at first but soon fuse, and fuse on the outside first because they grow more rapidly here. If the epithelium does not disappear before this fusion, many pathologic conditions result. The opposing surfaces of all the clefts fuse before the end of two months.

In 1881, His, studying exclusively human embryos and applying the reconstruction method, changed considerably the study of embryology. He found that the endodermal and ectodermal layers came in close apposition with at least only a very small amount of mesoderm or partition between the pharynx and the outside in the region of the clefts. It is not risking too much, he stated, to suppose that this membrane might tear, though, of course, this is not the rule. As one goes caudalward, the distance between the anterior ends of the arches becomes greater

so that at one time a triangular area is formed with its apex upward and was termed by His the mesobranchial field. During further growth, the arches telescope themselves from above down so that looking from the outside, the fourth is covered by the third and the third by the second. In this way, the third and fourth become encased in a cavity in the wall of the neck by the down growth of the second arch. This cavity His called the precervical sinus.

Von Mall (1887) agreed with His and Born. He found that in birds and higher vertebrates no breaking through of the branchial clefts took place, and that these clefts are always separated by a membrane composed of two epithelial layers. In view of the fact that these membranes are thin and delicate, it is possible, he stated, that they could rupture in the living embryo and thus give rise to many anomalies.

In 1888, Piersol came forth with his study of rabbit embryos. He came to the conclusion that the inner pharyngeal pouches are developed earlier and mature later than the outer arches and clefts. In mammals, the rule is, according to him, that the branchial clefts and pharyngeal pouches are separated by limiting membranes and that a tear can take place only in the second. In the first three pharyngeal pouches, two parts can be distinguished, the ventral horn and the posterior extremity, while, in the fourth, the parts cannot be differentiated. Out of the ventral horns, with the exception of the first, the tubular epithelial formations grow.

Foll, von Liessner, Kostschenke, Zimmerman, etc., took a stand opposite to that of His.

In 1884, Foll stated, after studying a 5-6 mm. human embryo, that the branchial clefts must be considered as continuous structures, even though delicate limiting membranes are interposed.

In 1889, von Liessner (studying chick and sheep embryos), concluded that patency of the branchial clefts does exist in all animals with certainty.

Hammer stated that in the 5 mm. embryo, the pharyngeal pouches are well developed and the second is perforated bilaterally at its base.

In summary, therefore, the prevailing theories concerning the origin of the so-called anomalies of the branchial cleft are:

1. The branchial clefts are responsible for cysts and fistulas. (*a*) A fistulous opening or rest may persist from any of the four clefts (von Heusinger, Bland-Sutton, von Cusset, His, etc.). (*b*) Only the second cleft can be at fault (Rabl, etc.). (*c*) The cervical sinus is responsible for the external aspect of the anomaly (von Ascherson, Kostaniecki, Milecki, His, etc.).

2. The thymic stalk and not only the clefts is responsible for their origin (Wenglowski).

EMBRYOLOGY

Early in the life of the embryo (within the first three weeks of intrauterine life), five bars are prominent in the region of the neck (figs. 1 and 2). These bars are spoken of as branchial arches and the depressions or slits between them as branchial grooves. Within the pharynx (fig. 3) there is a bar and groove opposite each external bar and groove. These structures are covered by ectoderm externally, endoderm internally and contain mesoderm between the two. The mesoderm between the external and internal grooves is absent or nearly so, so that the ectoderm and entoderm are practically adjacent, the membrane so formed

Fig. 1.—A 6.5 mm. embryo. *A* indicates lower extremity, *B* the tail, *C* the umbilical stalk, *D* the nose, *E* the eye, *F* the lower portion of the first branchial arch, *G* the second arch, *H* the third arch, *I* the heart and *K* the upper extremity. (From Wenglowski.)

in the first groove persisting to give rise to the tympanic membrane. These branchial arches and grooves are the forerunners of the gill bars and gill slits, respectively, in fishes, the adjacent ectoderm and entoderm in the depths of the groove having been absorbed to allow the pharynx to communicate with the outside. Hence the derivation of the word branchial (*βραγχια*; gills), meaning pertaining to or resembling the gills of a fish. In mammals, the branchial bars are the anlagen of definite and important structures of the face and neck. Though the grooves normally disappear, except the first, which becomes the external auditory

meatus and eustachian canal, they, by their embryologic nature, not altogether uncommonly persist by failure to occlude or by inclusion of epithelial and endothelial remnants and lay the foundation of the most interesting curiosities and pathologic problems that one encounters in the neck. It is, of course, with the latter that one wishes ultimately to deal, but they are often so complex that some knowledge of their embryologic development and relations is indispensable if one is to have accurate judgment in diagnosis and assurance in treatment.

Fig. 2.—A 6.5 mm. embryo. *A* indicates the branchial groove, *B* the mandibular portion of the first arch, *C* and *D* the nasofrontal process, *E* the eye, *F* the maxillary division of the second arch, *G* the second arch (operculum) and *H* the third arch (operculum). (From Wenglowski.)

The Arches.—The first arch is larger and grows more rapidly than the others, greatly deepening the oral fossa. It early divides into two portions. An upper, the maxillary, portion gives rise to the upper jaw, the lateral parts of the upper lip and the cheeks. The lower portion gives rise to the lower jaw, lower lip and region of the chin. This arch, as do the remaining arches, contains a rod of cartilage, which in this particular arch is known as Meckel's cartilage (figs. 3 and 7). This

cartilage extends from the symphysis menti to the tympanic cavity and lays the foundation of that part of the mandible which develops as intracartilaginous bone and which carries the incisor teeth (the remainder being intramembranous bone), the sphenomandibular ligament and two ossicles (malleus and incus).

Both ends of the rod of cartilage in the second arch ossify, the anterior end becoming the lesser cornu of the hyoid bone and the posterior end the styloid process, which fuses with the temporal bone. The intervening portion persists as the stylohyoid ligament.

That portion of the stapes other than the base is probably derived from a portion of the cartilage of the second arch, though its base develops as intramembranous bone. The portion of cartilage in the third arch becomes the greater horn of the hyoid bone, that from the two

Fig. 3.—A diagram to illustrate the branchial arches and grooves. *A* shows the cartilage, artery and nerve contained in the arch.

sides fusing through the body, which also develops from the third arch, and the whole fusing with the lesser cornua, later to form one piece.

The cartilage in the anterior portions of the fourth and fifth arches goes to make up the skeletal elements of the larynx and though it is thought that the greater part, at least, of the thyroid cartilage is developed from the fourth arch, the derivation of the remaining structures is not certain.

In addition to the structures mentioned, which are derived from the cartilage bars of the arches, one must consider the vascular, nervous and muscular content and also the epithelial bodies of the neck to which the arches give origin.

Vascular Content: One recalls that in the human embryo, six aortic arches appear on either side of the pharynx (fig. 4), taking their origin from the ventral aortic trunk and roots, coursing through the branchial arches and joining posteriorly the dorsal aortic roots. In the fishes and

larval amphibians, in which the arches and grooves develop into the gill bars and clefts, these aortic arches persist and develop into a ramifying network of small capillaries in the gill bars, which, when bathed by water circulating through the clefts, absorb its dissolved oxygen.

In mammals, however, marked changes occur early in embryonic life. The first two arches disappear completely (fig. 5), though the origin of the facial artery marks the anterior end of the first arch and the origin of the lingual marks the anterior end of the second. The third becomes the proximal portion of the internal carotid, the distal portion of the internal carotid being the cephalic remainder of the dorsal

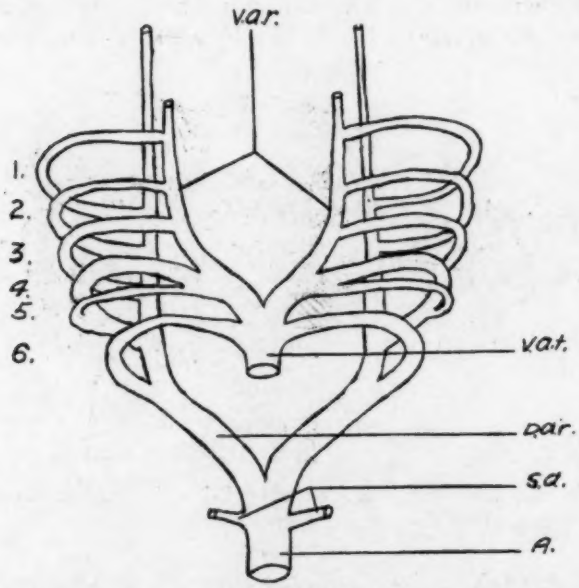

Fig. 4.—A diagram of the aortic arches of a mammal. *v a r* indicates the ventral aortic roots, *v a t* the ventral aortic trunk, *d a r* the dorsal aortic roots, *s a* the subclavian arteries and *A* the aorta. (From Bailey and Miller.)

aortic root from the junction of the third arch on. The fourth arch on the right and part of that dorsal aortic root becomes the right subclavian, while on the left the fourth arch remains as the arch of the aorta. The fifth and sixth arches disappear, with the reservation that the sixth on the left leading from the pulmonary artery (which has now been separated from the ventral aortic trunk by the septum aorticum) to the descending aorta persists for awhile as the ductus arteriosus (Botalli) and is the future ligamentum arteriosum. We will only mention here and refer to the fact in more detail later that a persistent second groove or remnant of the same would have to course superior to the internal

carotid and that from a third groove would have to course inferior to that artery. It is a well known fact that those fistulas the internal orifices of which are in the supratonsillar fossa and many branchial cysts lie in the fork of the internal and external carotids.

Nervous Content: The nerve of the first arch is the third division of the fifth (mandibular), while the second contains two, the facial (seventh) and the auditory (eighth). The third arch contains the glossopharyngeal (ninth), the fourth arch the superior laryngeal branch of the vagus and the fifth the inferior laryngeal.

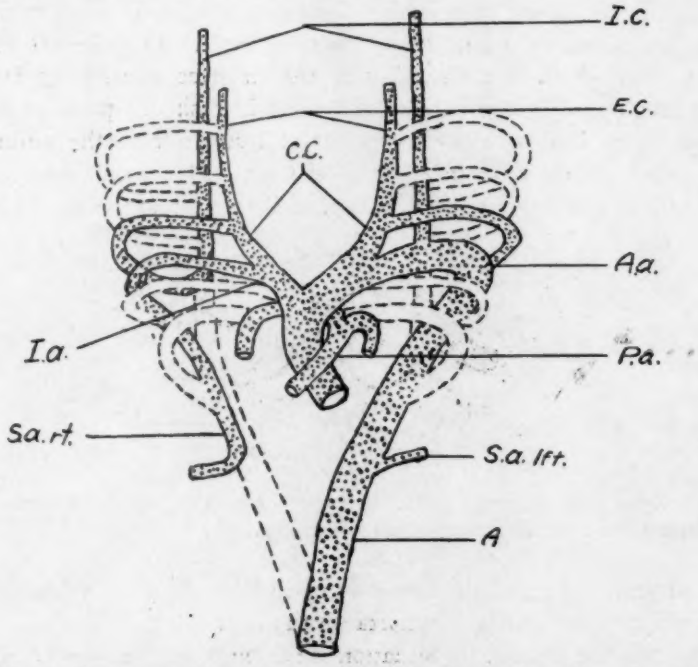

Fig. 5.—A diagram representing the changes in the aortic arches of a mammal. *I C* indicates the internal carotids, *E C* the external carotids, *C C* the common carotids, *I a* the innominate artery, *A a* the aortic arch, *P a* the pulmonary artery, *S a rt* the right subclavian, *S a lft* the left subclavian and *A* the aorta. (From Bailey and Miller.)

It is interesting to note in passing that, since the recurrent laryngeal is laid down anterior to the fourth arch, its forced migration caudalward by the subclavian on the right and the arch of the aorta on the left is accounted for.

Muscular Content: In view of the extensive and remote migration of the muscles about the face and neck, the problem of deducing their exact origin would be complex were it not for the fact that the nerves they carry with them serve as a key. Therefore, the muscles derived from the first arch (i.e., those innervated by the mandibular nerve) are

the muscles of mastication, the mylohyoid, anterior belly of the digastric, the tensor palati and tensor tympani. Those from the second arch (facial) are the stapedius, stylohyoid, posterior belly of the digastric, the platysma myoides and all the muscles of facial expression. Those of the third arch (ninth) are the stylopharyngeus and some of the soft palate muscles. The fourth (superior laryngeal) gives rise to some of the soft palate muscles, the constrictors of the pharynx and the cricothyroid, while the fifth (inferior laryngeal) is the origin of the intrinsic muscles of the larynx.

From the foregoing considerations one is able, if one assumes that either the second, third or fourth groove persists, to deduce the theoretical course of such a resulting fistula or cyst developing from the included rests. In the case of the second, it should pass between the external and internal carotid arteries and superior to the ninth nerve. The third should pass inferior to the internal carotid, lateral to the common carotid, inferior to the ninth and superior to the superior laryn-

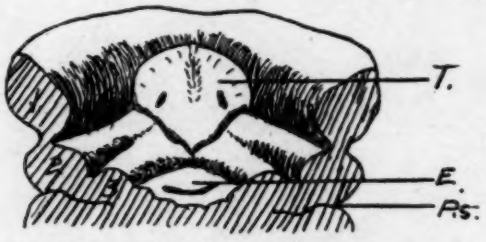

Fig. 6.—A diagram to illustrate the enclosure of the precervical sinus (*P S*). *T* indicates the tuberculum impar and *E* the epiglottis.

geal nerves. The fourth should course below the subclavian nerve on the right and the arch of the aorta on the left.

As a matter of fact, the situation is not quite this simple, for at about the fourth week of intra-uterine life the second arch grows outward and downward to meet the body wall, thereby enclosing the external aspect of the second, third and fourth grooves (fig. 6). The ectoderm lined pocket so formed is known as the cervical sinus and is probably responsible in diverse ways for the congenital anomalies. Thus, any of the following mechanisms might possibly obtain: 1. Any one of the grooves might remain as a fistula or a tract of included ectoderm and entoderm in pure form, having its origin and termination in its own normal embryologic location. 2. Any one of the grooves might persist with its normal embryologic origin on the pharyngeal side and by continuation in the cervical sinus have its external opening anywhere from the tip of the styloid process to the sternoclavicular joint. This might explain the fact that the internal orifice of a branchial fistula is almost always in the supratonsillar fossa (and, as will be seen later, undoubtedly originates

in the second groove), though its external orifice varies between the limits described. 3. Any groove might persist in whole or in part and connect with another groove through the cervical sinus.

Epithelial Bodies Derived from the Arches.—Tonsils: The tonsils (third month) arise in the region of the ventral part of the second inner branchial groove, are therefore entodermal in origin and grow as hollow buds into the mesenchymal tissue. Hollowed buds join the main bud, lymphoid cells wander in and by the third month after birth distinct lymph follicles with germinal centers are present.

Thymus: From the ventral part of the third branchial groove on each side (in the 6 mm. embryo), an entodermal evagination appears.

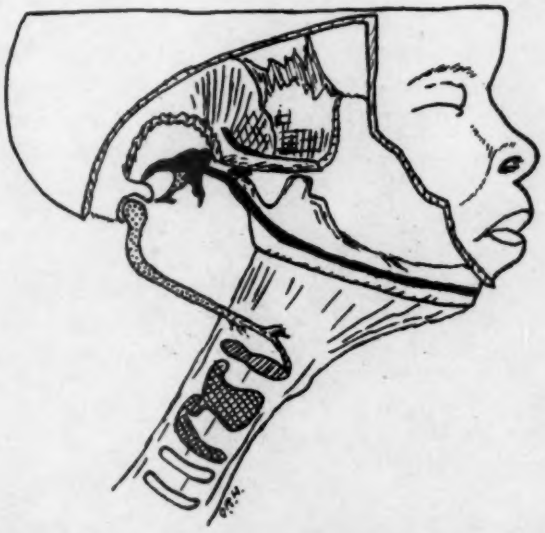

Fig. 7.—A diagram to illustrate the derivatives of the branchial arches. Complete black indicates the first arch (Meckel's cartilage), stipple the second arch, single cross hatch the third arch and double cross hatch the fourth and fifth arches.

This soon severs connection with its parent epithelium and grows caudalward as the thymic stalk, being ventral to the carotid arteries in the 16 mm. embryo and resting on the cephalic surface of the pericardium in the 29 mm. embryo, in which further differentiation of the bilobed thymus is carried out (fig. 8).

Thyroid Gland: In the 3-5 mm. embryo, at the point where the tuberculum impar and the two paired anlagen of the tongue join, there occurs an evagination of pharyngeal entoderm. From this, the site of the future foramen-caecum linguae, the mass of epithelium grows caudalward along the ventral surface of the larynx, giving rise to the thyroid duct. The terminal portion enlarges into the two lateral lobes remaining

connected by the isthmus, while the pyramidal process is a secondary growth from the isthmus, lateral lobes or distal thyroglossal duct.

CLASSIFICATION

It is obvious at once that branchial cysts may be classified from several points of view.

As to Position.—1. Branchial cysts may be superficial or deep (Bailey's classification). 2. They may be auricular or auditory, parotid, submaxillary, sublingual, pharyngeal or tracheal. This classification as to anatomic position alone not only is insufficient but fails to indicate the origin of the cyst, which is important.

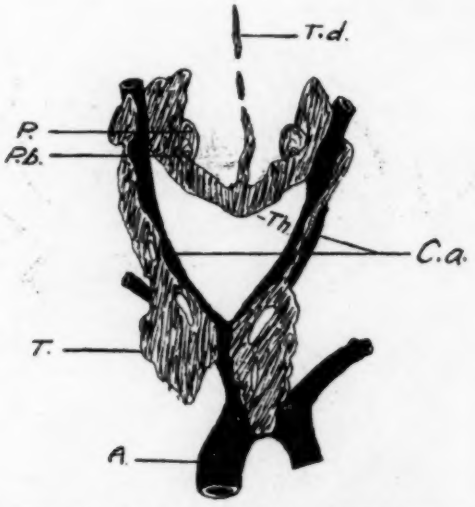

Fig. 8.—A diagram showing the branchial derivatives of a rabbit embryo of 16 mm. *T d* indicates the thyroglossal duct, *P* the parathyroids, *P b* the postbranchial bodies, *Th* the thyroid, *C a* the carotid arteries, *T* the thymus and *A* the aorta. (From Bailey and Miller.)

As to Cleft Giving Origin.—This classification is of little value, for it is a greatly disputed point whether more than one cleft is ever responsible for these congenital anomalies. Furthermore, in the case of cysts, it is often difficult to know which groove was responsible for its origin.

As to Contents.—1. Some branchial cysts are mucous cysts. These are usually the deep ones, lined by entodermal epithelium. Many of the so-called ranular cysts belong to this group, including the hour glass cyst of Roser and the congenital mucous cyst in the region of the base of the tongue and the sides of the larynx. 2. Some branchial cysts are atheromatous. These, at first having the appearance of dermoid cysts, are found to contain grumous material, which is entirely desquamated

epithelium without the elements of further differentiation, that is, hair, sweat glands, etc. 3. Other branchial cysts are serous. These have been known as "hydrocele colli" (Mannoir), "congenital hygroma of the neck" (Wernher), "congenital hydrocele of the neck" and "congenital cystic tumor of the neck" (Thomas Smith). These tumors appear as simple cysts lined with pavement epithelium and contain a limpid watery or tenacious fluid holding in suspension epithelial cells and cholesterol crystals.

Hematocysts of Branchial Clefts.—Of these there are two kinds: (1) those which are in communication with vessels and which can be emptied by pressure, and (2) those which are truly simple serous cysts with an admixture of blood, probably from minute hemorrhages in the wall of the cyst. These have been termed "haematocele colli" by Michaux. Senn mentioned that there might also be a true venous cyst in this location resulting from either a segment of vein completely constricted off at both ends or a saccular dilatation which has been constricted at its neck. Such a cyst, though in the region of the saphenous vein, was described by Paget in 1853.

Type of Lining Epithelium.—Lastly, branchial cysts may be classified according to the type of epithelium lining the wall and hence would resolve themselves into: (1) branchial cleft cysts with cylindrical epithelium, or branchial cleft mucous cysts; (2) branchial cleft cysts with pavement epithelium, or branchial cleft epidermoid cysts.

It seems that the latter classification by taking into full account their origin is by far the better.

FAMILIAL AND HEREDITARY TENDENCY

Though there are no reports on which to base an assertion of a hereditary tendency to branchial cysts, there have been several instances of such a tendency to fistulas. The following are reported in Virchow's archives (1861): mother and eight children; mother and son in three instances; mother and daughter; two sisters, a brother and a maternal grandmother; aunts in two instances.

In the familial instance we are reporting, a mother and three of her five children presented fistulas, but she is aware of no similar defect in other members of her family. Thus far, therefore, branchial fistulas may be familial and hereditary, and seem to be inherited only from the mother. No instances, so far as we know, have been reported of inheritance from the father.

CLINICAL FEATURES

This description of the clinical facts was drawn from a study of ninety cases taken from the literature and eighteen additional cases taken

from the archives of this hospital. An outline of the main facts brought out in these case reports is given at the end.

Cysts.—The average age of the persons in whom the ninety cases occurred was about 21 years; the oldest was 48 and the youngest 3 weeks at the time the patients were seen in the clinic. If, however, the average duration of the development of the cyst, which is about four years, is subtracted from these ages, the average age at onset of the cyst is 17 years. This applies only to those appearing some time after birth. In nine patients of this series (21 per cent), the cyst was present at birth. Though fistulas are not infrequently bilateral, we have found no report of a bilateral branchial cyst.

The patient comes to the clinic usually because of a painless swelling in the neck. Owing to its painless nature, the cyst often reaches the size of a hen's egg or lemon before it seriously attracts the patient's attention. In a small percentage of cases, however, the patient complains of pain (as in Thomson's [1] case, no. 29 in the accompanying table), particularly when the cyst swells and becomes tense. In respect to the swelling, many of the patients give a history of fluctuation in size. The cyst is often noted as becoming larger and more tense during the process of salivation, and indeed, as will be referred to later, Coplin [2] stated that in the case of branchial fistulas, stimulation of salivary secretion by citric acid or mastication usually stimulates secretion from the sinus even when it does not communicate with the alimentary canal. Though the cyst most frequently has an inconspicuous and insidious beginning, occasionally the patient traces it to some shock or injury, as in Jefferson's [3] case (no. 9) and as in Glover's [4] case (no. 27) of a patient who traced a slight swelling on the left side of the neck to an injury received while lifting heavy pipe in the oil fields. There was pain in his neck and it was stiff and sore for several days. He thought he had ruptured a muscle.

Though it is more frequent for the incomplete fistulas opening internally to give their first trouble after tonsillectomy, yet occasionally a deep cyst opening through a fistula internally will be brought to light through inflammation, suppuration or continued drainage after a tonsillectomy (Johnson,[5] case 3). Not infrequently does a cyst in conjunction with a fistula opening either externally or internally break through and drain copiously from time to time, obviously being of considerable annoyance. In one instance, the material issuing from an inner orifice was of foul

1. Thomson, J. W.: A Case of Branchial Cyst, Lancet **212**:76, 1927.
2. Coplin, W. M. L.: Branchial Cysts and Fistula, Proc. Path. Soc. Philadelphia **4**:109, 1900.
3. Jefferson, J. C.: Abnormalities of Branchial Origin, Clin. J. **45**:424, 1916.
4. Glover, G. E.: Branchial Cyst, M. Rec. & Ann. San Antonio **16**:171, 1922.
5. Johnson, J. A.: Branchial Cysts and Fistulae, Minnesota Med. **9**:514, 1926.

odor. Altogether too frequently to speak well of diagnostic acumen, the patient relates that the swelling has been aspirated many times (from four to fifteen times),[6] and occasionally incised and drained only to have it return again in every case. Such a procedure always sooner or later introduces infection. With the varying degrees of subsequent reaction, from mild inflammation to suppuration, the latter frequently with poor drainage, the lesion gives rise to pain and establishes a focus of pus and of toxins, which debilitate the victim greatly and cause a loss in weight. The inflammation also renders the wall of the cyst adherent to the surrounding structures, so that any subsequent attempt at dissection is made extremely difficult (Seed,[7] case 30). Not infrequently symptoms of pressure on contiguous structures are manifested. For example, Thomson [1] reported a case of cyst (no. 29 in the table), which swelled periodically and was associated with pain in the shoulder, dysphagia, dyspnea and a husky voice, which later advanced to complete aphonia, with fixation of the left vocal cord. These symptoms completely disappeared after extirpation of the cyst.

Though we are now discussing cysts, it is interesting to note Carp's [8] case of fistula (no. 8) in a boy of 5, who was troubled for two years by a dry, hacking cough, worse at night and when the fistula did not discharge. At operation, the fistulous tract was found to be adherent for a short distance to the vagus. The paroxysms of coughing completely disappeared after the operation. In Smith's [9] case (no. 43) of fistula in a child of three weeks, respiration and deglutition were markedly impaired. In one of our cases (E. E.), the fistula on the left opened into a cyst the size of which varied from that of a filbert to that of a hen's egg, and the patient would experience, in association with the swelling, nervousness, restlessness, hoarseness and palpitation of the heart. During the first attempt to probe the complete fistula on the right, it seemed that when a certain point was reached and before the probe was through the inner orifice, coughing, uneasiness and distress were stimulated. On the second attempt to probe the tract, sometime later, when we were unsuccessful on account of partial obliteration, the mere introduction of the filiform caused vomitus to well up in the throat.

The right side of the neck was involved in seventeen (35.5 per cent) and the left in twenty-one (64.5 per cent) of the cases. This proportion is about equally true for male and female taken separately.

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6. Bailey, H.: The Clinical Aspects of Branchial Cysts, Brit. J. Surg. **10**: 565, 1922-1923.

7. Seed, L.: Branchial Cyst, S. Clin. N. America **6**:1029, 1926.

8. Carp, Louis: Branchial Fistula; Its Clinical Relation to Irritation of the Vagus, Surg. Gynec. **42**:772, 1926.

9. Smith, Thomas: Congenital Cystic Tumor, St. Bartholomew's Hosp. Rep. **2**:16, 1866.

On examination, one finds a regular hemispherical tumor, the skin surface over which appears smooth. The size may be from that which is just detectable to that which fills the whole side of the neck. It is often described as being the size of a filbert, a walnut, a hen's egg or an orange, etc. It may be large enough to protrude in the floor of the mouth as in the case reported by Senn [10] (no. 40). Treves [11] reported a case in an infant (no. 44), in which the whole side of the neck and the thorax on that side down to the umbilicus was involved. The cyst may be located about the pinna and anywhere from this region to the junction of the sternocleidomastoid and the clavicle, and even may extend somewhat lower than this, so that it is partly substernal, the swelling pointing in the suprasternal notch. Those cysts that are located in the neck bear a definite and constant relation to the sternocleidomastoid muscle. It can be said with impunity that if they are of any considerable size, they extend, at least in part, under this muscle and this is one of the most dependable clinical features of this type of cyst. Submaxillary cysts are, of course, excepted. Frequently the fibers of the muscle will be thinned out owing to long continued pressure from the cyst and one might get the false impression that the muscle belly goes through the tumor. The true relationship in many cases, however, may be easily brought out by merely having the patient attempt to rotate his head while the examiner holds the chin, so as to place the sternocleidomastoid muscle on the stretch, and at the same time palpating it. As the cysts grow larger to accommodate their accumulated secretion and contents, they dissect their way, of course, along the planes of least resistance and the direction that they take depends considerably on the depth of the cyst. In regard to their depth, Bailey [6] has classified them as (1) those lying just beneath the skin; (2) those extending deeper and attached to the internal jugular; (3) those passing between and straddling the bifurcation of the common carotid, and (4) those of the mucous or columnar cell type lying entirely posterior to the common carotid next to the pharynx. The larger and more superficial ones may extend toward the midline and bring up the question of thyroglossal duct cyst, but they practically always are eccentric enough to preclude being the latter. It seems to have been the rule that the cysts taking origin in the neck may dissect as high as possible under the sternomastoid muscle and therefore behind the ear but seldom dissect over the jaw and anterior to the ear as may hygromas. This point was brought to bear in a recent questionable diagnosis at this hospital.

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10. Senn, N.: Branchial Cysts of the Neck, J. A. M. A. **3**:197 (Aug. 23) 1884.

11. Dissection of a Congenital Hydrocele of the Neck, Virchows u. Hirsche Jahresbericht **1**:280, 1882; quoted by Pranesk.

The uncomplicated cyst is most frequently obviously fluctuant and rather soft, though the so-called atheromatous or dermoid types may be rather firm and nonfluctuant. The latter may be lobulated, rendering their differentiation in diagnosis from tuberculous glands especially difficult. This difficulty is increased if secondary infection has taken place and more especially if there is an externally opening fistula through which the puslike or grumous material periodically escapes. If the latter condition is present, an examination of the fistulous orifice with a hand lens will be enlightening, provided the inflammatory process has not completely destroyed the anatomic definitive. The uncomplicated cyst has been described as having a multilocular feel, but this is not common. Although one cannot get the sensation by feeling through the overlying structure, yet, at operation, when feeling through only the thin wall of the cyst, the sensation one gets is much like that when feeling lung. The cyst wall is not adherent to the overlying structures, but feels to be so deeply, and this assumption is, in most cases, proved to be true at operation. Uncomplicated branchial cysts are not painful, except in rare cases owing to pressure, and are not tender.

Secondary infection, of course, changes the picture completely. All the signs of inflammation obtain, with adherence to the surrounding structures, pain, tenderness, fever, leukocytosis, etc.

Fistula.—In the series of sixty-two cases of branchial fistulas, 53 per cent occurred in females and 47 per cent in males. Fifty-one and eight tenths per cent were on the right side, 31.4 per cent were on the left and 16.8 per cent were bilateral. One interesting case reported in Virchow's archives (1861) presented multiple small openings on the right at the junction of the sternocleidomastoid muscle with the clavicle. This is the only case of multiple unilateral fistulas that we were able to find record of. It may be noted that the right and left incidence here is the reverse of that for cysts.

In the case of fistula, the feature that brings the patient to the hospital is practically always drainage, which may be periodic. It may consist of clear, tenacious mucus (said not to be of the chemical character of saliva) or thin, milky and puslike.

True fistulas practically always exist from birth and may be of such capillary dimensions as often to escape notice. Obviously, the incomplete fistulas that open only internally may never come to attention unless they are large enough to fill with food or unless they become infected and drain habitually, a condition which, as mentioned, is not infrequently initiated by tonsillectomy. A fistula opening into the trachea may give rise to air sacs and present the features of what has been termed "aerial goiter," "aerial bronchocele," "tracheocele" and "hernia of the trachea" (Coplin;[2] Eldridge[12]). Eldridge described such

12. Eldridge, S.: Am. J. M. Sc. **78**:70, 1879.

a condition in a patient 26 years of age—an air cyst on each side of the lower part of the pharynx and the upper part of the trachea, which grew larger on expiration and increased the circumference of the neck from 40.5 to 49 cm. The external orifice may be situated about the pinna, or in the neck always just anterior to the sternomastoid muscle.

Often there is a small areola of brownish discoloration about the external orifice and occasionally a small bit of tissue, which is invariably found to contain a piece of cartilage. These are known as "supernumerary" or "cervical auricles."

By picking up the skin about the orifice, one can feel the cordlike tract extending into the neck. By traction, it prevents the orifice from being lifted as high as the surrounding skin.

Mention has been made of such symptoms and signs as pallor, uneasiness, palpitation of the heart, coughing and vomiting on probing the fistula, which can be adequately accounted for by the proximity of a portion of the tract to the vagus.

Among unusual cases that have been reported, the following are of passing interest:

Lesser reported a patient in 1840 (Virchow's archives), who traveled with a show and made his livelihood by passing a curved knitting needle into the pharyngeal orifice of a fistula and extracting it from the external orifice.

Ascherson presented a patient who, though having originally only one external orifice, later developed three, the fistula closing and opening in a new place each time.

Von Berg showed a patient in whom the external opening was in the incisura thyroidea and communicated with the middle ear. When the external orifice closed, material would discharge from the ear, while injections made in the external opening gave rise to pain in the middle ear.

Branchial cleft fistulas have also been described as occurring in cats and dogs.[13]

DIAGNOSIS

In arriving at a diagnosis of branchial cleft cyst, one should have in mind that it is usually a uniform, painless, fluctuant, semifixed, nontranslucent tumor with a characteristic relationship to the sternocleidomastoid muscle and without tenderness. The differential diagnosis involves the following:

Adenitis.—Nontuberculous adenitis usually involves more than one gland and not infrequently both sides, and tenderness is the rule. The offending focus may be found in the tonsils or the teeth. Tuberculous

13. Heusinger: Halsfiemenfisteln des Menschen und Thiere, Deutsche Ztschr. f. Thiermed. **2**:1, 1876.

adenitis is the most difficult to rule out. Of course, no effort should be spared on behalf of the patient to rule this out, for the stigma of tuberculosis in a young person is of considerable gravity. The fact that aspirated material may resemble tuberculous pus closely and be free from pyogenic organisms tends only to confirm the physician's original diagnosis of tuberculosis, unless he is aware of the possibility of its being a branchial cyst. The likelihood of the lesion's becoming infected and draining for an indefinite period also supports his delusion.

Though the atheromatous branchial cyst filled with grumous cheese-like material is glandular and firm and, if secondarily infected, is adherent to the surrounding structures and tender, yet in tuberculous adenitis it is more frequent to find other swollen glands or a group of matted glands. It is of some diagnostic importance that branchial cysts seem more common in the white race and tuberculous adenitis more common in the colored race. A cyst associated with an external fistula might be deceiving, but, as mentioned, a careful examination of the orifice of the fistula can be enlightening.

Cystic Hygroma.—Translucency is the most important differential feature and can be said itself to be diagnostic.

Thyroglossal Duct Cyst.—Such cuts are practically always in the midline and are so attached to the thyroid gland or trachea that they move forcibly during deglutition. Microscopically, it is frequently impossible to make the distinction.

Venous Hemangiomas.—These growths decrease in size on pressure, though one must be sure the contents of the tumor have not merely been squeezed into another compartment of the same.

Retropharyngeal Abscesses.—Such abscesses are painful and may exhibit swelling and tenderness on only a cursory examination of the pharynx.

Lipomas.—Lipomas, as well as other solid tumors, do not yield fluid material on aspiration.

For diagnosis, the demonstration of cholesterol crystals in the aspirated material of the cyst is helpful, though dermoid cysts produce this substance also.

Fistulas are sometimes injected with colored substances, such as methylene-blue (methylthionine chloride, U. S. P.), which, if the fistulas are complete, will issue from the internal orifice into the mouth. Bismuth paste has been injected and x-ray photographs obtained (cases 28 and 31). They have also been probed with small flexible filiforms, bristles of hair, etc. In one of our cases (fig. 9), we were able completely to pass an x-ray ureteral catheter and obtained roentgenograms, which were studied for the direction and curvature and relation of the tract to the hyoid bone.

PATHOLOGY

In removing a branchial cyst, one often finds it attached at one pole to a fistulous tract, which most usually extends toward the tonsillar fossa. This tract often diminishes down to such fine caliber that it cannot be completely dissected. The wall of the cyst is usually smooth, though it may vary in thickness from that of tissue paper to 0.25 cm. It has been mentioned that one of these had the feel of lung, which was accounted for probably by numerous interlacing, tough, fibrous strands seen on the inner aspect of the wall, which were at first suspected of being hair.

Grossly, the content of the cyst is usually a thick, transparent, mucoid, sticky substance in those lined by columnar epithelium, while it

Fig. 9.—An x-ray picture of a patient with a ureteral catheter in a fistula. The markers designate the external orifice and the internal orifice in the tonsillar fossa.

is opaque, watery and milky or cheeselike and of all the consistencies between the two in those lined by squamous epithelium. The fluid content often can pass through a hypodermic needle, and though it may bear a striking resemblance to tuberculous pus, if some of it is placed in water, the shimmering lipoid substance will be evident floating on the surface.

The cellular content has been found (Coplin [2]) to be as follows: (1) large cells resembling squamous epithelial cells, 93.7 per cent; (2) polymorphonuclear leukocytes, 1.8 per cent; (3) erythrocytes, 0.5 per cent, and (4) mononuclear leukocytes and unidentified cells, 4 per cent.

Gurlt, quoted by Senn,[10] mentioned a great similarity between the contents of these cysts and the contents of ovarian cysts. He mentioned

the careful chemical examination made by Hoppe, which was as follows: 1.3953 Gm. of atheromatous material contained 0.2225 alcoholic extractive matter, 0.1235 ethereal extractive matter, 0.2005 aqueous extractive matter and 0.8555 insoluble matter. In these substances, he found leucine, tyrosine, cholesterol, myelin, palmitin and stearin.

Histologically, the walls of the cyst present many interesting features. The most usual observation is a well organized, stratified, squamous epithelium resting on a relatively thick band of lymphoid

Fig. 10—A photomicrograph of the wall of a branchial cyst to illustrate a stratified epithelium resting on a connective tissue base without the interposition of a band of lymphoid tissue. (Case 51, S. R.)

tissue, while beyond this is connective tissue, which may contain varying amounts of smooth and striated muscle (most likely extracapsular), and which is usually infiltrated to some extent with the elements of chronic inflammation—round cells, mononuclears and an occasional eosinophil leukocyte. The zone of lymphoid tissue may be entirely absent, the epithelium resting immediately on connective tissue (fig. 10). Again, it may be scant or abundant and with distinct and well formed lymph follicles (fig. 11). The epithelial layer, though in most cases

composed of stratified squamous cells, is now and then columnar with or without cilia (fig. 12).

One of our sections exhibited both the stratified and the columnar epithelium, while one showed well formed mucous glands just beneath the squamous epithelium. The differentiation from dermoids and other anomalies of earlier ontogenetic origin and their close simulation of the commoner epidermoids lacking hair, sweat glands,, etc., has been mentioned.

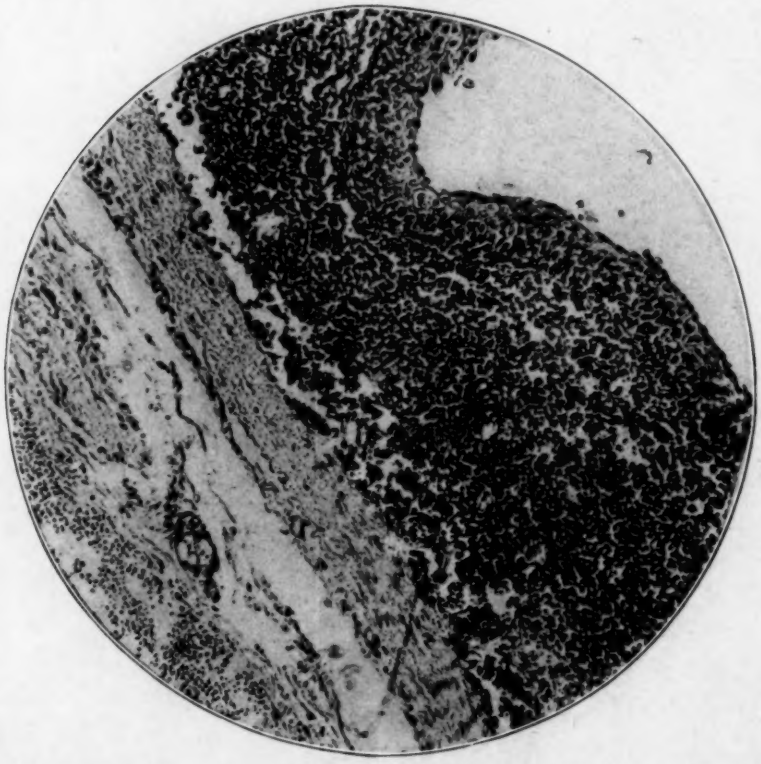

Fig. 11.—A photomicrograph of the wall of a branchial cyst to illustrate a stratified epithelium on a dense lymphoid tissue base. (Case 45, L. P.)

The stratified squamous epithelial layer appears like that of normal skin. There are, however, no papillae, except rudimentary ones, and the various layers are not discernible. The stratum germinativum or rete malpighii may be evident forming a row of cells with their oblong nuclei set perpendicular to the base, often containing considerable pigment and showing, as well as do many of their offspring, definite prickle cell characters. The nuclei are large, well defined and reticulated and contain one or more chromatin masses. They take the hematoxylin stain well and often maintain these characters well out to the stratum

corneum, where they suddenly cornify and are sloughed away. The cytoplasm stains darkly, appears finely granular and in places, owing to the prickled borders, is easily outlined. In some cases, the cells soon lose their original characteristics and become vacuolated long before reaching the stratum corneum. Though it has been mentioned (Coplin[2]) that the lymphatic layer is not especially vascular, we find the contrary to be true in our sections, many of them showing numerous vessels. It is true, however, that most of these also exhibit the elements of chronic inflammation.

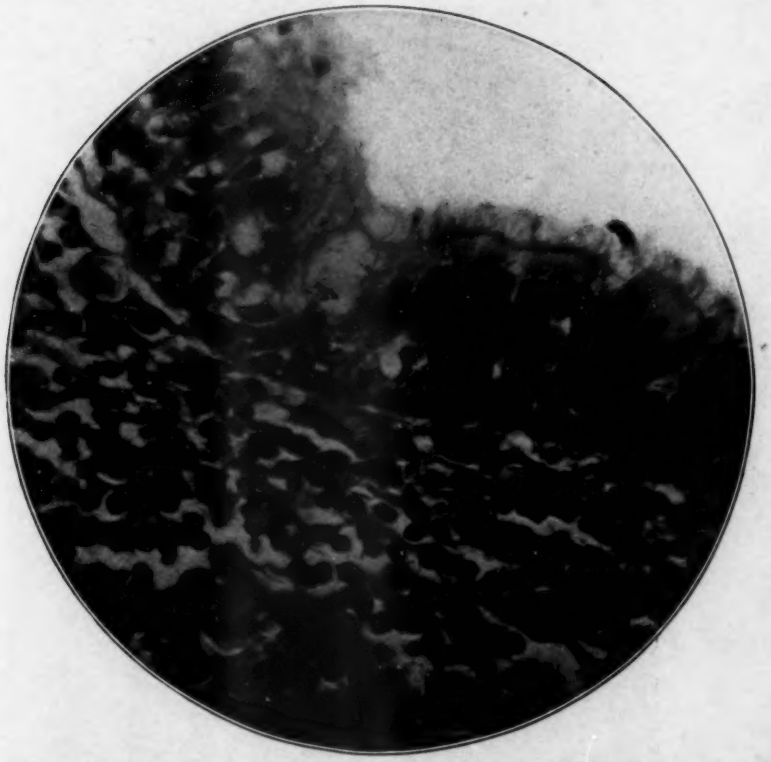

Fig. 12.—A photomicrograph of the wall of a cyst to illustrate the ciliated columnar epithelium on a rather dense lymphoid tissue base. (Case 48, N. H.)

There are certain anatomic and histologic features of branchial cysts and fistulas that make their origin solely from branchial clefts more convincing than an origin from thymic stalk, as maintained by Wenglowski.[14] The latter believed that cysts and fistulous tracts lined with stratified squamous epithelium are ectodermal and must undoubtedly be of cervical sinus (Halssinus) origin, while the anomalies

14. Wenglowski, R.: Ueber die Halsfisteln und cysten, Arch. f. klin. Chir. **100**:789, 1913.

and portions of the anomaly that are entodermal are of thymic stalk origin. He felt that the dense lymphoid tissue with sometimes definite lymphoid follicles and occasionally what appear to be Hassal bodies in the walls of cysts and fistulas is strong evidence of thymic origin. It

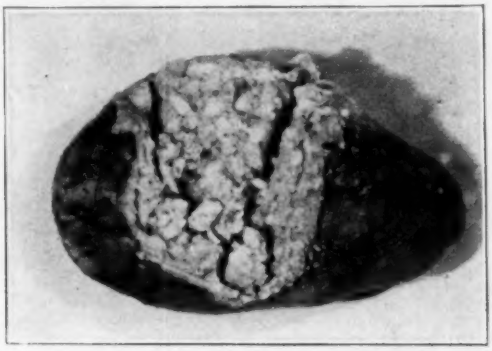

Fig. 13.—A photograph (natural size) of an atheromatous branchial cyst removed from the right submaxillary region of V. H. (fig. 14). The cyst has been ruptured exposing the grumous contents of desquamated epithelial cells and cholesterol crystals. The wall was lined with stratified squamous epithelium.

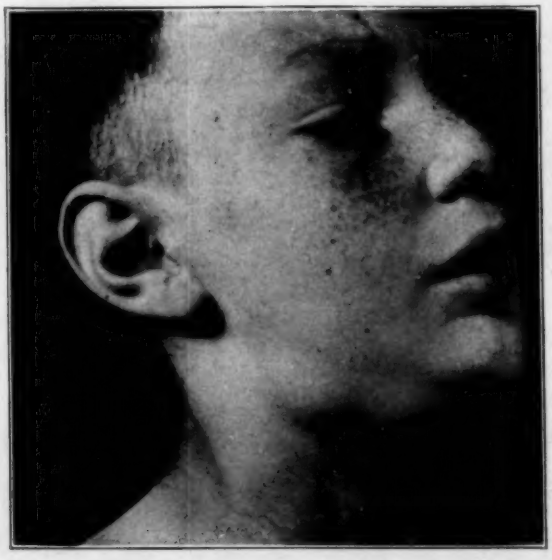

Fig. 14.—Patient V. H. with an atheromatous branchial cyst in the submaxillary region of the right side. A photograph of the gross specimen is shown in figure 13.

seems, however, that, in view of the fact that cysts lined wholly by prickle cell stratified epithelium, and therefore of undoubted ectodermal origin, are commonly found exhibiting the dense band of lymphoid

tissue, the latter cannot be used to bear witness of thymic origin. For the thymic anlage cannot have had to do with such a cyst. Further, the cysts located as high as the region of the tip of the mastoid present the same histologic characteristics, and there is certainly no reasonable proximity of such a structure to the region of the assumed remains of the thymic stalk.

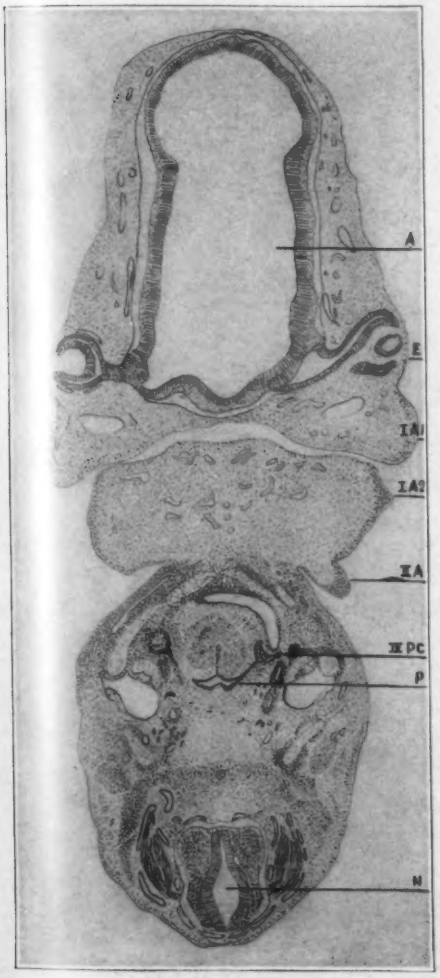

Figure 15

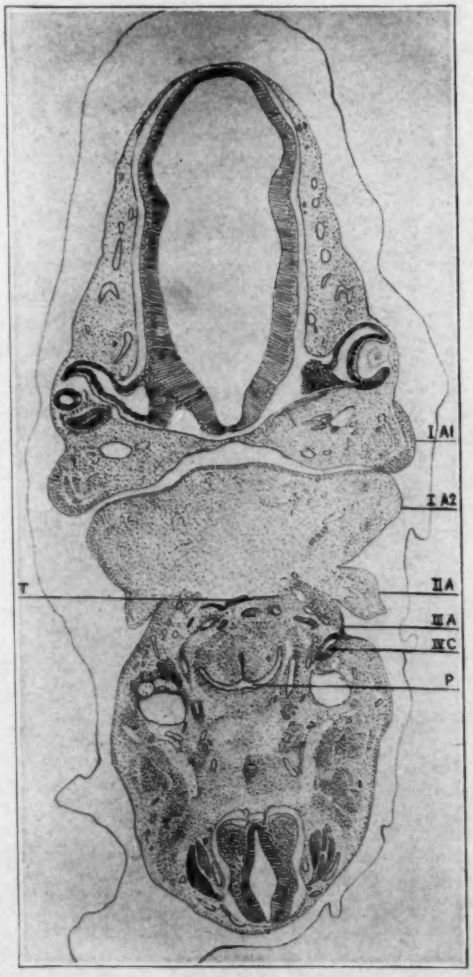

Figure 16

Fig. 15.—*A* indicates the brain cavity, *E* the eye, *IA1* the superior division of the first branchial arch, *IA2* the inferior division of the first branchial arch, *IIA* the second branchial arch, *IVPC* the fourth pharyngeal cleft, *P* the pharyngeal canal and *N* the neural canal.

Fig. 16.—*IA1* indicates the superior division of the first branchial arch; *IA2*, the inferior division of the first branchial arch; *IIA*, the second arch; *T*, the thyroid anlage (the thyroglossal duct has disappeared at this state); *IIIA*, the third arch; *IVC*, the fourth cleft and *P*, the pharyngeal canal.

Wenglowski brought out that some of the external fistulas point downward instead of upward. One would expect this occasionally to be true, but so far as we were able to ascertain, no such fistula extended far enough to be convincing in this regard. In patient E. E. (case 58), the filiform, when passed into the left orifice, turned downward

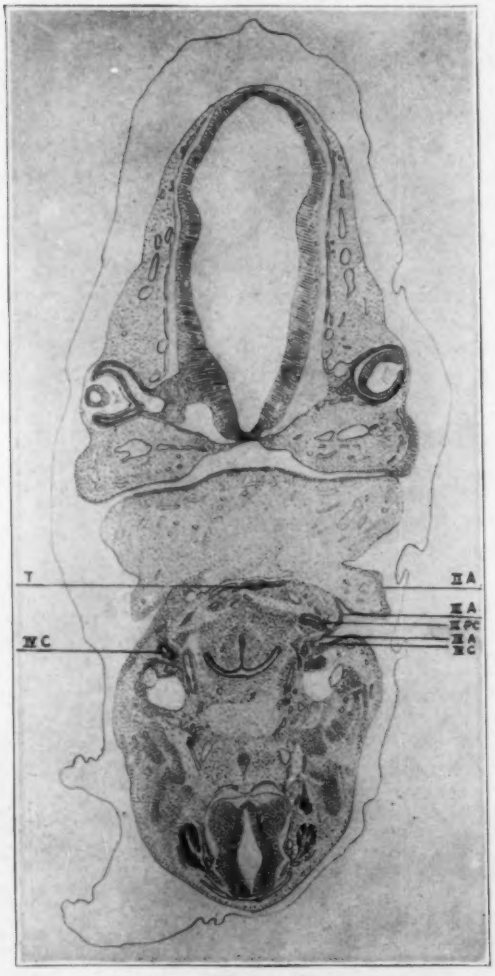

Figure 17

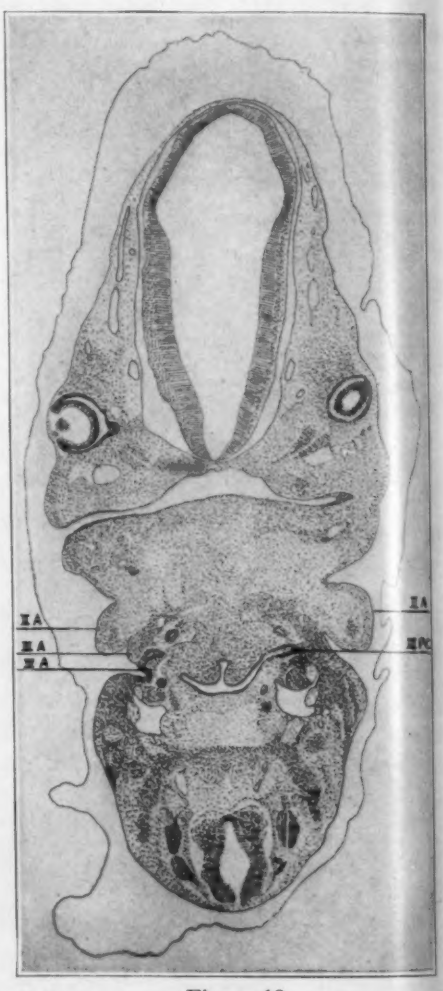

Figure 18

Fig. 17.—*IIA* indicates the second arch; *T*, the thyroid anlage; *IIIA*, the third arch; *IIIPC*, the third pharyngeal cleft; *IVA*, the fourth arch and *IVC*, the fourth cleft. The second arch is seen here to be growing downward to meet the body wall and inclosing the third and fourth arches forming what is known as the cervical sinus (Halssinus).

Fig. 18.—*IIA* indicates the second arch; *IIIPC*, the third pharyngeal cleft, now joining the pharyngeal canal; *IIIA*, the third arch and *IVA*, the fourth arch and cervical sinus shown on the right.

about one-half inch (1.27 cm.), but then turned up suddenly and joined a definite cyst. Also, if the remains of a thymic stalk played a rôle, cystic structures should occur anywhere from the final resting place of the thymus to the region of its origin. Langebeck, quoted by Senn,

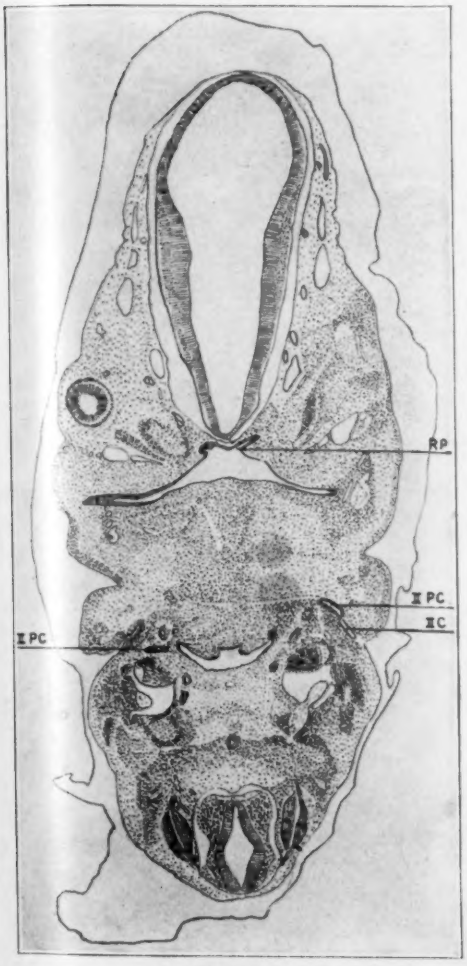

Figure 19

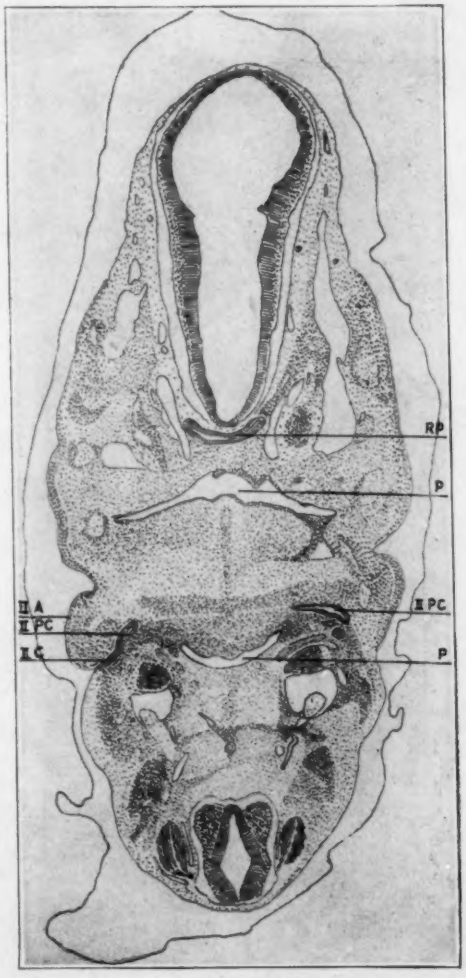

Figure 20

Fig. 19.—*RP* indicates Rathke's pouch; *IIPC*, the second pharyngeal cleft and *IIC*, the second cleft.

Fig. 20.—*RP* indicates Rathke's pouch; *P*, the pharyngeal canal; *IIPC*, the second pharyngeal cleft; *IIA*, the second arch and *IIC*, the second cleft.

stated that branchial cysts are frequently attached to the greater horn of the hyoid bone or to the thyrohyoid ligaments, localities that plainly indicate that they originated from remnants of former branchial clefts.

So far as fistulas are concerned, the third groove, being embryologic like the second, would seem to have the same chances of persisting, though so far as the external and complete fistulas are concerned, their tracts are practically always reported as coursing between the external and internal carotid arteries and having exit in the supratonsillar fossa and therefore must be of second groove origin. We are in poor position

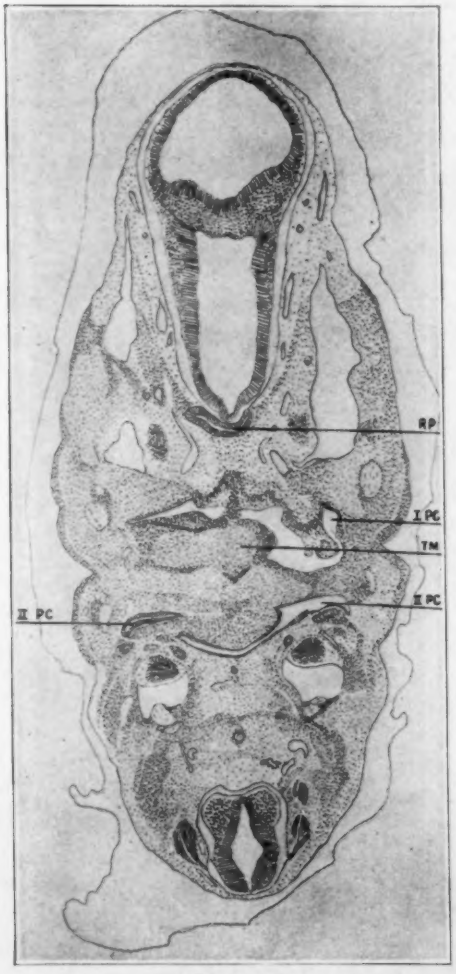

Figure 21

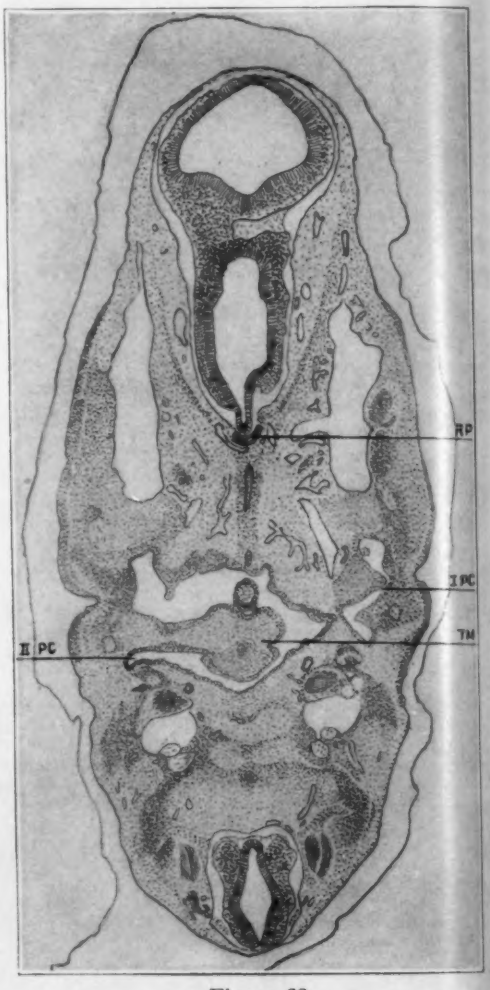

Figure 22

Fig. 21.—*RP* indicates Rathke's pouch; *IPC*, the first pharyngeal cleft on its way to meet the first outer cleft shown later, which forms the eustachean tube; *TM*, the tongue mass; *IIPC*, the second pharyngeal cleft.

Fig. 22.—*RP* indicates Rathke's pouch; *IPC*, the first pharyngeal cleft; *TM*, the tongue mass and *IIPC*, the second pharyngeal cleft. The external first clefts are shown on both sides here, which will be met soon by the pharyngeal or internal first clefts. The first external clefts form the external auditory meatus.

to draw conclusions about incomplete internal fistulas. These must be far more common than one suspects, for it is not reasonable to suppose that they would ever be discovered if they remain relatively undisturbing to the patient. It is often extremely difficult to locate the internal orifices.

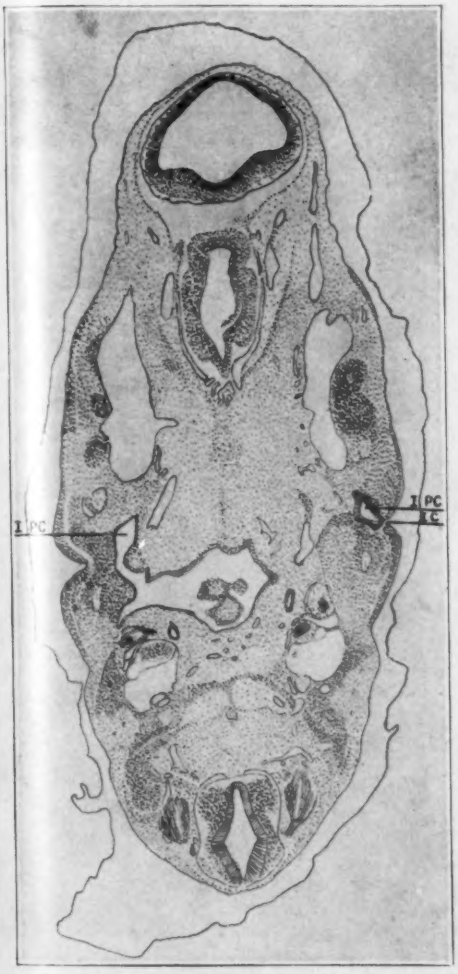

Figure 23

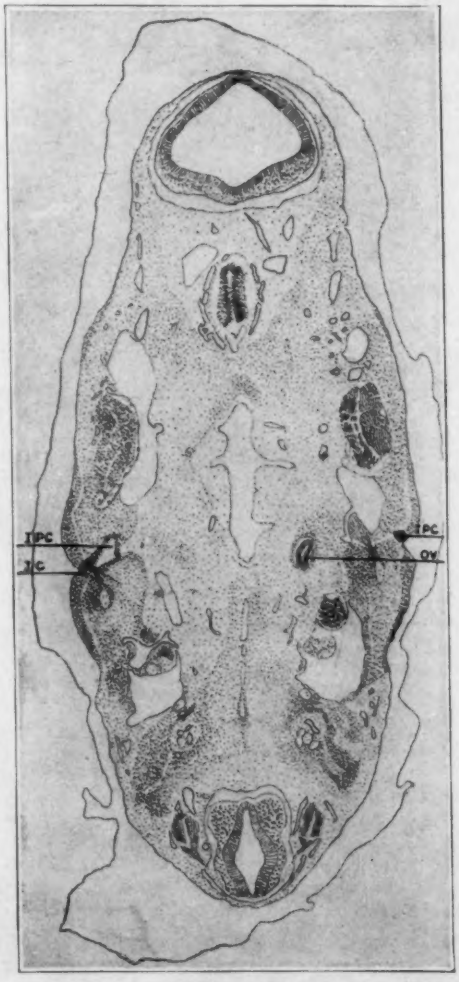

Figure 24

Fig. 23.—*IPC* indicates the first pharyngeal cleft and *IC*, the first (external) cleft. The membrane between *IPC* and *IC* forms the tympanic membrane.

Fig. 24.—*IPC* indicates the first pharyngeal cleft; *IC*, the first (external) cleft and *OV*, the otic vesicle.

We have no report of a fistulous tract coursing inferior to the right subclavian artery or aortic arch, and believe, therefore, that the fourth groove is practically never responsible for such structures.

So far as the branchial grooves ever being normally patent is concerned, we find no indication of it in the serial sections of a 7.5 mm. human embryo (figs. 15 to 25). Owing to the manner in which serial sections are cut, one might get the impression of a patency from the pharynx to the outside but on following the true pharyngeal side

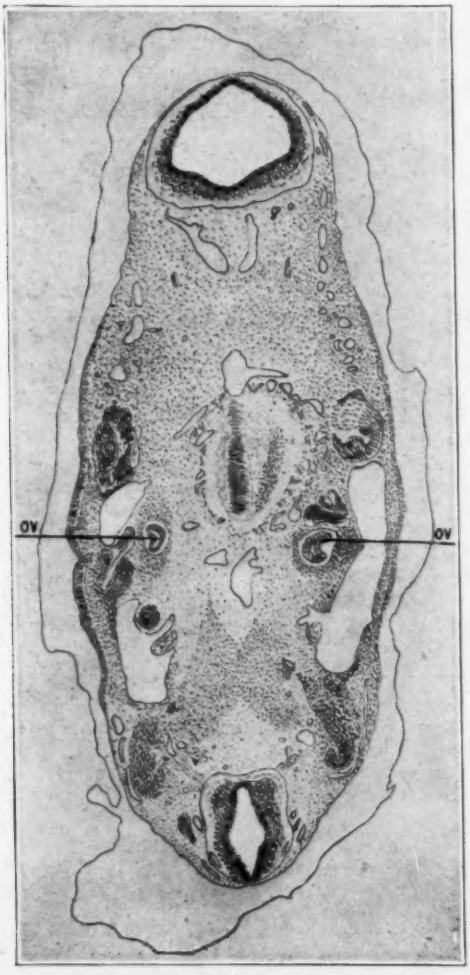

Fig. 25.—*OV* indicates the otic vesicle.

of the groove this is seen not to be the case (i.e., the manner in which the fourth external cleft is cut on the left in figures 15 and 16 and on the right in figures 16 and 17 might be deceiving in this regard).

It seems far fetched to assume a tear of the groove membrane in early embryonic life as the cause of fistulas. In assuming this, one is practically limited to trauma, and it is hard to believe that in so small

an embryo, so well protected, trauma could be of just the extent to establish a fistulous communication between the pharynx and the outside. Even if such an accident occurred, there would be no logical reason for its persistence. Again, it cannot be supposed that cysts, the origin of which must be the same as that of fistulas, are established by tears of the groove membrane in a neck that has never presented the stigmas of external or internal fistulas.

There has been little contributed on the subject of the origin or existence of aural fistulas. The anomaly has usually been lightly passed over with the assumption that it originates in the first branchial groove.

Fig. 26.—Patient E. E. (case 58), with bilateral cervical fistulas and an aural fistula on the left. A urethral filiform is shown entering the external orifice and coming through the mouth having made its exit in the supratonsillar fossa.

It is difficult, however, to attribute it to such an origin when, as is known, the first groove normally persists in the formation of the external auditory meatus and the groove membrane in the formation of the tympanic membrane. It has been also suggested that fistulas, as well as cysts, in this region are the result of epithelial inclusions or of anomalous development of the several tubercles that go to form the pinna. The fistula was blind in all of our familial cases, was only an eighth to a quarter of an inch (from 0.3 cm. to 0.6 cm.) in depth and had never been the site of a discharge.

Histologically, these anomalies present none of the structures of advanced differentiation such as hair follicles or sudoriferous glands, though in one of our sections well formed mucous glands were contained in the cyst wall just beneath the lining epithelium. The explanation of this difference from teratomas and dermoids rests on the fact that the inclusions are made after the three germinal tissues have well differentiated. Branchial cysts are indeed, therefore, not greatly different from any of the commoner epidermoid cysts, i.e., the epidermoid cysts about the face and palms of the hands resulting from epithelial inclusions or bits of epithelium buried by trauma and subsequently continuing their active growth and desquamation. In the latter there is often, too, a marked lymphoid infiltration in the cyst wall, but so far as we know, no well defined lymphoid follicles.

Fig. 27.—Patient E. E., showing symmetry of the cervical fistulas.

TREATMENT

The treatment of branchial cleft cysts and fistulas may be dismissed with one consideration—complete excision of the epithelial wall. This may be simple for superficial cysts, but technically extremely difficult for deep cysts and fistulas. We feel that it is not advisable to inject the cyst with methylene blue at operation owing to the marked disadvantage of having the operative field stained with the dye should the cyst rupture. If the operator is anxious to determine a possible communication with an internally opening fistula, the disadvantage might be obviated by washing the dye out of the cyst, thereby leaving only its wall stained. The relation of branchial anomalies to the vital structures of the neck makes their dissection a tedious one, and it should be attempted only by those who are acquainted with the anatomic minutiae

of the neck; for, obviously, if the dissection is not complete, the technical difficulty of a second operation is increased many fold.

Most of the commoner corrosive substances have frequently been injected into the cysts, but are invariably without effect.

Fig. 28.—Patient E. E., illustrating the external orifices of symmetrical bilateral cervical fistulas and an aural fistula on the left. The right cervical fistula was complete, while the left led to a cyst, which fluctuated from the size of a filbert to that of a hen's egg.

Figures 15 to 25 were selected from 200 camera lucida drawings prepared by Dr. H. J. Prentiss from serial sections made of a 7.5 mm. human embryo. The sections were 10 microns thick and the drawings

Fig. 29.—Patient C. E., showing a cervical fistula and a probe in the aural fistula on the right.

Fig. 30.—Patient W. E., showing bilateral cervical fistulas and probes in bilateral aural fistulas.

made from alternate sections. This stage of embryonic life is about the optimum for the illustration of the branchial apparatus. The sections were cut in such a way that the two halves are not symmetrical. Also, it should be remembered that due to the marked flexion of the embryo ventrally, sections taken from ventrum to dorsum will progress caudalward in the head region and cephalicward in the tail region. The sections shown in figures 15 to 25 progress ventrodorsally.

REPORT OF CASES

The following group of cases, which occurred in a mother, two daughters and a son, present familial branchial cleft fistulas:

CASE 1.—E. E., a colored woman, aged 28, entered the hospital with a complaint of frequent sore throat for two or three years past, a lump in the left side of the neck of four years' duration and a discharging sinus on each side of the neck. The family history and the social history were essentially without bearing. She had

Fig. 31.—Patient B. E., showing a cervical fistula and a probe in the aural fistula on the right.

five children, living and well, and had had no miscarriages. She said that she had never had any signs or symptoms of venereal infection. She had noticed the sinuses in the neck from birth, and from time to time (about three or four times a year) had observed that they seemed to swell slightly just at the point of exit and that in a short time they broke down discharging a few drops of whitish, translucent material. She felt that the one on the right opened into the throat, for occasionally a foul tasting substance seemed to be ejected on the right side of the mouth. For the past four years, she had noticed a swelling in the left side of the neck, which varied from the size of a filbert to that of a hen's egg. The fluctuation in the size of this swelling had no relation to mastication. When the mass swelled to its maximal size, she experienced nervousness and palpitation of the heart. There seemed to be also some difficulty in talking; that is, her voice seemed hoarse. In addition to these symptoms, she experienced pain in the region of the left shoulder, which she herself attributed to the lump in the neck because it was present only when this lump was swollen to its maximal size.

Summary of Data in Sixty-One Cases of Branchial Cleft Cysts and Fistulas

Author	Age, Years	Race	Sex	Fistula (Side of Neck Involved)	Cyst (Side of Neck Involved)	Duration	Location in Neck	Hereditary Tendency	Diagnosis	Operation	Result	Remarks	Pathology
1. Crow, I. N.: J. Iowa M. Soc. **15**: 524, 1925	48	W	M	Right	.....	?	Tonsillar fossa	0	Correct	+	Well	Cleared white particles from throat at times; fistula from 2 to 3 cm. in length, emptying into tonsillar fossa	Stratified squamous epithelium; lymphoid near basement membrane
2. Whitacre, H.J.: Ann. Surg. **37**: 56, 1903	5	W	F	Right	.....	Since birth	Lower third	0	Correct	+	Well		
3. Johnson [5].....	24	..	F	.....	Left	1.5 yr.	Middle third	0	Correct	+	Well	Began after tonsillectomy.....	Stratified squamous epithelium
4. Johnson [5].....	14	..	M	.....	Right	Since birth	Lower third	0	Correct	+	Well	Discharging fistula at birth healed after six years; after four years more, became infected; between external and internal carotids	Pus was aspirated at one time, but at operation cyst contained 2 ounces of semigelatinous fluid; stratified squamous epithelium
5. Johnson [5].....	17	..	F	.....	Right	Since birth	Lower third	0	Correct	+	Well	Between external and internal carotids; no communication with pharynx	Pear shaped cyst; inner wall smooth and filled with mucm
6. Johnson [5].....	21	..	M	Left	.....	Since birth	Lower third	0	Correct	+	Well	Between external and internal carotids; no communication with pharynx; discharged seropurulent material for seven years; healed fourteen years, then broke down	Cyst 5 by 1 cm., lined with stratified epithelium
7. Johnson [5].....	7 mo.	..	M	Left	.....	Since birth	Lower third	0	Correct	0	Well	Had remained closed for one year when last heard from	
8. Carp [6].....	5	..	M	Right	.....	Since birth	Lower third	0	Correct	+	Well	Healed from time to time; first discharged at 2 years; paroxysms of coughing ceased after operation; between carotid artery and vagus	Tract 3 by 1 cm., containing mucoid material; lined by squamous epithelium on lymphoid tissue
9. Jefferson [3].....	18	..	F	.....	Right	6 wk.	Middle third	0	Correct	+	Well	Supposed to have followed trauma	
10. Jefferson [3].....	47	..	F	.....	Left	5 yr.	Middle third	0	Correct	+	?	Tender and brawny; pus drained and later cyst excised; between external and internal carotids and connected to pharyngeal wall by stalk	
11. Coplin [2].....	36	..	F	.....	Right	3 yr.	Upper third	0	Lipoma ?	+	Well		Sac 7 cm. in long diameter; squamous epithelium on lymphoid base; contained many one, two and three nucleated epithelial cells and a few polymorphonuclears

12. Bevan, A. D.: S. Clinics, Chicago **4**: 1, 1920	19	..	M	.....	Left	2 yr.	Upper third	0	Correct	+	Well	Size of lemon, adherent to jugular vein	Squamous epithelium; scant lymphoid base
13. Bailey [6].....	17	..	M	.....	Lower third	2 yr.	.....	0	Tuberculous adenitis	+	?	Aspirated seven times; then cyst size of tangerine shelled out	
14. Bailey [6].....	20	..	F	.....	Left	12 yr.	.....	0	Tuberculous adenitis	+	?	Aspirated five times; later, cyst size of egg shelled out	
15. Bailey [6].....	27	..	F	.....	Lower third	2 yr.	.....	0	Tuberculous adenitis	+	?	Aspirated fifteen times; later, cyst size of orange shelled out	
16. Bailey [6].....	35	..	M	.....	Lower third	18 mo.	.....	0	Tuberculous adenitis	+	?	Incised and drained; later, large cyst dissected out	
17. Bailey [6].....	20	..	F	.....	Lower third	14 mo.	.....	0	Tuberculous adenitis	+	?	Aspirated five times; pus sterile; given tuberculin; later, egg size cyst excised	
18. Bailey [6].....	20	..	M	.....	Left	14 mo.	Middle third	0	Tuberculous adenitis	+	?	Aspirated four times; later, orange size cyst dissected out	
19. Bailey [6].....	20	..	F	.....	Right	10 yr. 4 mo.	Middle third	0	Correct	+	?	Cyst size of orange	
20. Bailey [6].....	18	..	M	.....	Left	.....	Middle third	0	Correct	+	?	Cyst size of orange	
21. Bailey [6].....	34	..	F	.....	Right	Life	Middle third	0	Correct	+	?	Cyst size of orange	
22. Bailey [6].....	40	..	M	.....	Right	1 yr.	Middle third	0	Correct	+	?	Size of egg	
23. Bailey [6].....	19	..	M	.....	Left	3 yr.	Upper third	0	Correct	+	?	Size of tangerine orange	
24. Bailey [6].....	21	..	M	.....	Right	1 yr.	Middle third	0	Correct	+	?	Size of orange	
25. Bailey [6].....	46	..	F	.....	Right	2.3 yr.	Upper third	0	Parotid tumor	+	?	Hazelnut size	
26. Becker, J.: Beitr. z. klin. Chir. **134**: 470, 1925	24	..	M	Right	.....	Life	Lower third	0	Correct	0	?	Methylene blue came out by tonsil	
27. Glover [4].....	37	..	M	.....	Left	9 yr.	Middle third	0	?	+	?	Patient thought tumor initiated when he was injured in oil field	
28. Meyer, H. W.: Am. J. Surg. **11**: 121, 1926	27	..	M	Left	.... .	Life	Lower third	0	Correct	+	?	Bismuth paste injected; x-ray picture revealed complete fistula, with exit in tonsillar fossa; foul-smelling fluid could be expressed; the tract passed above the greater cornu of hyoid bone	
29. Thomson [1].....	35	..	M	.....	Left	3 yr.	Middle third	0	Tuberculous adenitis	+	Well	Periodic swelling causing pain in the shoulder, husky voice, later dysphagia, dyspnea and aphonia; did not go between external and internal carotids, but into pharynx	Cylindrical and cuboidal epithelium with scant lymphoid tissue base

Summary of Data in Sixty-One Cases of Branchial Cleft Cysts and Fistulas—Continued

Author	Age, Years	Race	Sex	Fistula (Side of Neck Involved)	Cyst (Side of Neck Involved)	Duration	Location in Neck	Hereditary Tendency	Diagnosis	Operation	Result	Remarks	Pathology
30. Seed [7].....	16	..	F	.....	Left	16 mo.	Middle third	0	Tuberculous adenitis	+	?	Extended to bifurcation of carotid; patient was debilitated and had lost much weight	Stratified squamous epithelium
31. Eddowes.....	30	W	F	Right	.....	Birth	Lower third	Grandmother	Correct	+	Good	Injected with bismuth paste; discharged white to yellow material; extended from lower two-thirds of neck to lateral wall of pharynx; incomplete	
32. Gilman.....	13	W	F	Right	.....	Birth	Lower third	0	Correct	+	Good	Iodine and silver nitrate had been injected without avail; after being dissected, left jugular vein completely stripped and the styloid process, submaxillary gland and right wall of pharynx became visible in the wound; the cyst was adherent to the carotid sheath	Contained semisolid gruellike material with cholesterol crystals
33. Langenbeck.....	17	W	F	.....	Left	Since 12	Middle and lower thirds	0	Correct	+	Good	Adherent to carotid sheath, it had been only partially dissected out at first and then drained; later completely dissected out	
34. Langenbeck.....	20	W	F	.....	Left	Since 9	Middle third	0	Correct	+	Good	Reached to cervical vertebra posteriorly	Contents revealed epithelial cells in a state of fatty degeneration
35. Schede.....	15	W	M	.....	?	18 mo.	?	0	Correct	+	Good	Adherent to carotid sheath; size of hen's egg; had been irritated by injections; indicates that injection of irritating substances is responsible for adherence	Contents revealed epithelial cells in a state of fatty degeneration
36. Schede.....	22	W	F	.....	Left	Since 11	Upper third	0	Correct	—	?	Attached to sheath of carotid artery	Creamy, yellowish fluid, containing free fat and epithelia; walls studded with sebaceous follicles especially toward the carotid artery; portion attached to sheath of vessels contained plate of cartilage which resembled the cartilage of the ear; hence he called it "auricular teratoma"

37. Virchow.....	34	W	F	.....	?	Since 14	Upper third	0	Correct	+	Good	When cyst was out, revealed esophagus, lateral wall of pharynx, carotid artery in sheath and great horn of hyoid bone; jugular vein was ruptured	Contents showed flat epithelial cells, cholesterol crystals, fat granules and a mass of debris, the products of epithelial degeneration; wall lined with stratified epithelium
38. Senn [10]..	36	W	F	.....	Right	1 yr.	Upper third	0	.....	—	.....	Located over bifurcation of common carotid; pulsated distinctly and made a pressure against floor of mouth and larynx; size of hen's egg	
39. Senn [10].....	22	W	F	.....	Right	1 yr.	Upper third	0	Correct	+	Good	Protruded in floor of mouth and pushed tongue against palate	"Microscopic examination revealed it to be of branchial origin"
40. Senn [10]...	25	W	F	.....	?	4 yr.	Under chin	0	Correct	?			
41. Wernher.....	Child	W	F	.....	Left	Since birth	Lower, middle, upper thirds	0	.....	Drained	?		
42. Smith, T.[9].. ...	3 mo.	W	M	.....	Right	Since birth	Behind ear	0	.....	0	?	Respiration and deglutition impaired; child died of smallpox, and autopsy revealed that tumor had disappeared after having drained six months	
43. Smith, T.[9].....	3 wk.	W	F	.....	Left	Since birth	Lower, middle, upper thirds	0	.....	0	?	Tumor got smaller until it disappeared	Dark-brown, reddish, syrupy fluid escaped through rupture
44. Treves [11].....	Infant	?	F	.....	?	?	?	0	Correct	+	Good	Occupied whole side of neck and upper part of thorax on the same side, extending to umbilicus; contained one large and numerous smaller cysts; "corresponded to region of second branchial cleft"	
45. Hyndman, I. P.	18	W	F	.....	Left	16 mo.	Upper third	0	Correct	+	Good	Cyst had formerly been incised and drained; size of hen's egg	Stratified squamous epithelium; much lymphoid infiltration, with lymph follicles
46. P. B.	22	W	F	.....	Left	11 yr.	Middle third	0	Correct	+	Good	Had been incised several times and a transparent mucoid material obtained; was partially excised, except proximal tract, which caused recurrence; the latter was painful	Stratified epithelium; lymphoid infiltration
47. L. H.	24	W	F	.....	Right	3 yr.	Middle third	0	Correct	+	Good	Size of lemon; extended part way under mandible	Stratified squamous epithelium
48. N. H.	6	W	M	.....	Left	5.5 yr.	Lower third	0	Correct	+	Good	Hyoid bone felt in fossa after excision; cordlike tract at upper pole	Stratified and columnar epithelium; chronically inflamed

Summary of Data in Sixty-One Cases of Branchial Cleft Cysts and Fistulas—Continued

	Author	Age, Years	Race	Sex	Fistula (Side of Neck Involved)	Cyst (Side of Neck Involved)	Duration	Location in Neck	Hereditary Tendency	Diagnosis	Operation	Result	Remarks	Pathology
49.	E. E.	32	W	F	.....	Left	9 mo.	Upper third	0	Correct	+	Good	Size of hickory nut	Stratified squamous epithelium with well formed lymph follicles; contained cheesy material
50.	R. J.	32	W	F	.....	Left	?	Middle, upper thirds	0	Correct	+	Good	Size of egg; at operation, it lay on the vascular sheath	Stratified epithelium with much lymphoid infiltration; contained mass of epithelial cells and cholesterol crystals
51.	S. R.	22	W	F	.....	Right ear	2 yr.	Upper third	0	Correct	+	Good	Size of hazelnut; attached to small pedicle	Stratified epithelium and remnants of epithelial structures in wall
52.	M. W.	21	W	M	.....	Right	3 mo.	Upper third	0	Correct	+	Good	About size of hen's egg; fluctuated in size; never painful	Cyst wall trabeculated with grayish fibrous strands; walls contained lymphoid follicles
53.	R. B.	19	W	F	.....	Left	3 mo.	Upper third	0	Correct	+	Good	Contained a milky fluid; extended deeply to jugular foramen; never painful	
54.	D. R.	18	W	M	.....	Left	6 mo.	Upper third	0	Tuberculosis	+	Good	Reached to base of skull from angle of jaw and contained creamy fluid	
55.	K. E.	20	W	F	.....	Left	2 mo.	Middle third	0	Tuberculosis	+	Good	Size of hazelnut; two firm glands attached; contained milky fluid	Stratified squamous epithelium with lymphoid tissue and follicles in wall
56.	L. B.	14	W	F	Right	Right	10 yr.	Lower third	0	?	+	.....	Size of golf ball; contained yellowish caseous material	Stratified squamous epithelium on a lymphoid tissue base
57.	L. G.	30	W	M	.....	Right	2 yr.	Upper third	0	?	+	Good	Keloid developed in scar; would swell and discharge yellowish material; complete fistula discovered at operation	Columnar epithelium on a base of fibrous tissue with a few lymphocytes
58.	E. E.	28	C	..	Left and right	.....	.....	Middle third	See case reports					
59.	C. E.	3	C	M	Right	.....	.....	Middle third	See case reports					
60.	W. E.	5	C	M	Left and right	.....	.....	Middle third	See case reports					
61.	B. E.	6 wk.	C	..	Right	.....	.....	Middle third	See case reports					

The forty-six cases of branchial cleft fistulas reported in Virchow's Archiv fur pathologische anatomie und Physiologie for 1861 were not included in this table.

On examination, the external fistulas were seen, as shown in the photograph. Aside from a marked hypertrophy of the tonsils and some hypertension, the results of the physical examination were essentially negative. The tonsils were so large that the internal orifices of the fistulas could not be seen until after the probe was passed. A urethral filiform catheter was passed with little difficulty through the right fistula; it emerged in the supratonsillar fossa. The filiform entered the fistula on the left and pointed down about one-half inch (1.27 cm.), then turned abruptly up and entered apparently into a somewhat firm lump on the left side of the neck. The internal orifice was not found on this side. The filiform on the right was followed by an x-ray catheter, after the passage of which stereo x-ray pictures were obtained. After the catheter had been introduced one half to two thirds of the way in the fistula on the right side, uneasiness and coughing were induced. Feeling that the stiff catheter would distort the fistulous tract to some extent, we later decided to inject iodized poppy seed oil 40 per cent. But after two weeks the fistula had become obliterated to such an extent that it was impossible to inject the iodized oil. This patient also had a fistula, just anterior to the left ear, which was about one fourth of an inch deep (0.63 cm.). There was no discharge from the fistula. In fact, it had never been brought to the patient's attention.

CASE 2.—C. E., a colored boy, aged 3, had a fistula on the right side of the neck and a right aural fistula, as shown in figure 29. Otherwise he was normal. The cervical fistula, on pressure, emitted a drop of clear, mucoid, tenacious fluid. The discharge from this fistula to the time of writing had been slight.

CASE 3.—W. E., a colored boy, aged 4 years, 8 months, had bilateral cervical fistulas and bilateral aural fistulas, as shown in figure 30. The results of the physical examination were otherwise negative. One of the cervical fistulas, to the time of writing, had discharged several times without pain or swelling.

CASE 4.—B. E., a colored girl, aged 6 weeks, had cervical fistula and aural fistula on the right (fig. 31). She presented no other abnormalities. On pressure, a small amount of clear, mucoid fluid could be expressed.

The two remaining children did not present branchial anomalies.

CONCLUSIONS

1. Branchial cleft anomalies (cysts and fistulas) result from a failure of absorption of the included ectodermal and entodermal epithelium that is buried during the growth and fusion of the branchial arches in early embryonic life. This is most probably the sole explanation, the thymic stalk playing no rôle.

2. Branchial cysts, in truth, are epidermoid cysts of the neck whose parent epithelium was buried during the development of the branchial apparatus. Their characters are more varied, of course, than those of the commoner epidermoid or inclusion cysts owing to the activity of entodermal or ectodermal epithelium or both. Many of the submaxillary cysts and so-called ranulae are of branchial origin.

3. Branchial fistulas may be familial and hereditary, and they seem to be inherited through the mother only.

4. More attention should be given to the possibility that a tumor in the neck is of branchial origin, especially in view of their frequent simulation of tuberculous glands and their occurrence in early youth. This is enjoined even more by the fact that results from treatment are in proportion to the completeness with which the epithelial wall is excised.

5. An adequate and simple classification of the cysts would be (*a*) branchial cleft epidermoid cysts and (*b*) branchial cleft mucous cysts.

LYMPHATIC DRAINAGE FROM THE PERITONEAL CAVITY IN THE DOG *

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Accurate data concerning the extent and the routes of drainage from the abdominal cavity of patients with gastro-intestinal lesions are of extreme importance. Pulmonary involvement, so commonly associated with abdominal operations, is a sequela which may be explained by the lymphatic continuity of the two regions. Many experiments have been made in an effort to determine the site and rate of absorption from the peritoneal cavity and the routes through which such materials are removed. Cunningham [1] completely reviewed the literature which concerns these various phases of the problem.

In the hitherto published accounts of experimental studies on the method of lymphatic drainage following peritoneal absorption, more careful attention has been given to the cytologic problems than to the lymphatic paths which are involved; also to the manner in which the foreign particles within the peritoneal cavity get into the lymph stream, whether free and independent or in phagocytic cells, than to the distribution of the lymph channels throughout the diaphragm, mediastinum and pulmonary regions. In the process of absorption from the peritoneal cavity, the lymphatic tracts of the diaphragm are most extensively involved. From this muscular partition the lymph may flow either through the anterior mediastinum and its associated lymph nodes or, to a lesser degree, into the thoracic duct and thence to the venous circulation at the jugular subclavian confluence.

Our interest in the problem of peritoneal absorption grew out of a desire for more accurate knowledge concerning the lymphatic system of the diaphragm and its relation to the lymphatics of the lungs in the leading experimental animal, the dog. Excellent descriptions, adequately illustrated, of the lymphatic distribution in this region in man are available in standard textbooks on anatomy, but adequate descriptions of the system in this region in the dog are not available. Sisson [2] con-

* Submitted for publication, Oct. 4, 1928.

1. Cunningham, R. S.: The Physiology of the Serous Membranes, Physiol. Rev. **6**:242, 1926.

2. Sisson, Septimus: The Anatomy of the Domestic Animals, ed. 2, Philadelphia, W. B. Saunders Company, 1914, p. 930.

tributed little if any data on the lymphatics of the diaphragm and their relations to the lung. Since the dog is being used more and more as an experimental animal, it is increasingly important to obtain such data. Furthermore, the exact relationship of the lymphatic system to the vascular system is inadequately understood. The fact that normal lymph so often contains large numbers of erythrocytes leads one to question the generally accepted statements that the lymphatic system is a closed one. Accordingly, it may be worth while to seek further detail bearing on this most important but little understood lymphatic organization.

Many excellent reports of experimental studies on the absorption of particulate matter from the peritoneal cavity, with special reference to the reactions of free phagocytic cells, are available. The mode of transfer of such particulate matter into the diaphragmatic lymphatics, whether through definite stomata as originally advanced by von Recklinghausen,[3] or directly through the cell bodies, are among the questions discussed. MacCallum [4] denied the existence of stomata and attributed absorption to the activity of free phagocytic cells. The pumping action of the respiratory movements facilitated the passage of these phagocytic cells into the lymph stream, although in the absence of such macrophages the mechanical factors involved literally forced free particles through the wall of the lymph channel.

Various injecting mediums have been used in the earlier studies of peritoneal absorption. Buxton and Torrey [5] recovered nucleated blood cells from the mediastinal lymph nodes fifteen minutes after their intraperitoneal injection into a guinea-pig. Bolton [6] employed lampblack, bacteria and nucleated blood cells in his study of absorption, and concluded that the drainage is a purely mechanical process, the force being supplied by respiratory movements, and that the particles pass between the epithelial cells of the diaphragm. Bolton briefly commented on the position of the lymph channels which drain the diaphragm. Cunningham [7] injected into the peritoneal cavity of cats a solution consisting of washed nucleated blood cells, unfiltered carbon particles and lamp-

3. Von Recklinghausen, F. T.: Zur Fettresorption, Virchows Arch. f. path. Anat. **26**:172, 1863.

4. MacCallum, W. G.: On the Relation of the Lymphatics to the Peritoneal Cavity in the Diaphragm and the Mechanism of Absorption of Granular Materials from the Peritoneum, Anat. Anz. Centralbl. **23**:157, 1903.

5. Buxton, B. H., and Torrey, J. C.: Absorption from the Peritoneal Cavity, J. M. Research **15**:5, 1906.

6. Bolton, Charles: Absorption from the Peritoneal Cavity, J. Path. & Bact. **24**:429, 1921.

7. Cunningham, R. S.: Studies in Absorption from Serous Cavities: IV. On the Passage of Blood Cells and Granules of Different Sizes Through the Walls of the Lymphatics of the Diaphragm, Am. J. Phys. **62**:248. 1922.

black granules in isotonic sodium chloride solution. In three minutes all three types of material had reached the anterior mediastinal lymph nodes, but were all free in the sinuses. The foreign particles had not been phagocytosed in their transfer to the lymph stream, but moved freely into the surface cells of the diaphragm. In Cunningham's study on the absorption from the peritoneal cavity in the fetus, respiratory movements seem intimately correlated with the extent of absorption. Such movements of the diaphragm literally force the particles into the cells where they are carried through the endothelium into the lymph duct.

Cunningham,[1] in a complete review of the physiology of serous membranes, concluded that at least a large proportion of the particulate matter passes through, and not between, the cells in the course of its absorption into the diaphragmatic lymphatics from the peritoneal cavity. He further concluded that one significant factor in the absorption of particulate matter through the mesothelial cells is by means of the pressure of the diaphragm on the intra-abdominal viscera. Florey,[8] in studies on the rabbit, concluded that foreign particles pass from the peritoneal cavity between the mesothelial cells of the diaphragm into the lymphatic trunks and not directly through the cytoplasm of these cells. Florey supported the original thesis of MacCallum (1903), but did not substantiate the observations of Cunningham (1922). Poynter[9] has conducted extensive experiments on the absorption of true solutions, colloids, bacteria and particulate matter from the peritoneal cavity, and concluded that lymphatic channels, except those related to the diaphragm, play but an insignificant part in the absorptive process. The venules of the omentum are shown by Poynter to be the more important avenue, because various colloidal particles have been recovered from the portal blood and the liver. The mesenteric lymphatics, the receptaculum chyli and the thoracic duct were never found to participate in the drainage from the peritoneal cavity. More recently Brown,[10] in an experimental investigation to determine the value of lymphaticostomy, concluded that in rabbits the main path of absorption from the peritoneal cavity is by the thoracic duct, but in dogs and cats this route plays only a subsidiary part. He concluded that lymphaticostomy cannot be recommended in cases of acute peritonitis. Although we have observed the mode of absorption and the nature of the particulate matter in the lymph stream in a number of instances,

8. Florey, Howard: Reactions of, and Absorption by Lymphatics, with Special Reference to Those of Diaphragm, Brit. J. Exper. Path. **8**:479, 1927.

9. Poynter, C. W. M.: Peritoneal Absorption, Proc. Staff Meetings, Mayo Clinic, **2**:263, 1927.

10. Brown, K. P.: Peritoneal Lymphatic Absorption: An Experimental Investigation to Determine the Value of Lymphaticostomy, Brit. J. Surg. **15**:538, 1928.

we shall not report them here, since this paper is restricted entirely to a consideration of the lymphatic channels which are involved in the drainage of particulate matter from the peritoneal cavity in the dog.

METHODS OF STUDY

Various types of particulate matter have been used from time to time in the study of peritoneal absorption. Florey used hemoglobin, india ink, and suspensions of carmine and cream, but his best results were obtained by using finely particulate graphite, first described by Drinker and Churchill,[11] and known as "Hydrokollag 300." Higgins and Murphy[12] modified somewhat the method of making the suspension described by Drinker, and greatly reduced the interval required to perfect the final product. When made correctly and thoroughly centrifuged the final preparation is a homogenous mass of particles many times smaller than erythrocytes. The preparation may be injected directly into the blood stream of mammals without causing any reaction, and thus it provides an admirable means for the study of the reticulo-endothelial system. We have used the graphite preparation repeatedly for studies on the von Kupffer cells of the liver, the reticular cells of the spleen and the phagocytic cells of serous cavities, and have found it an entirely satisfactory injection mass.

OBSERVATIONS

If a given quantity of the final graphite preparation, the amount varying with the size of the animal, is introduced into the peritoneal cavity of a dog, the lymphatics of the diaphragm are soon visible with the injection particles. In periods ranging from ten minutes to an hour and a half the lymphatic channels on the pleural surface of the diaphragm are completely delineated (fig. 1). Muscular activity appeared to hasten the absorptive process for, in animals exercised during the interval following the intraperitoneal injection, the lymphatics were more readily and more completely filled. Massage of the wall of the abdomen and chest further facilitated absorption, and since the process seemed greatly retarded in animals suffering with distemper or other pulmonary disability, respiration played a significant part in the removal of such particulate matter from the peritoneal cavity.

One hour after the peritoneal injection of from 40 to 50 cc. of the graphite preparation, the lymphatic channels on the pleural surface of the diaphragm were well distended and their distribution could be

11. Drinker, C. K., and Churchill, E. D.: A Graphite Suspension for Intravital Injection of Capillaries, Proc. Roy. Soc. London **101**:462. 1927.

12. Higgins, G. M., and Murphy, G. T.: The v. Kupffer Cell in Common Laboratory Animals, Anat. Rec. **40**:15, 1928.

readily traced. Although some variation in the extent and distribution of the lymphatic pattern maintained in different animals, yet in the twenty-five dogs which comprised our series, the more characteristic picture is that shown in figure 1. It must be recalled that the lymphatics here illustrated are those functionally active in such normal absorptive processes, delineated only by the graphite and not by the injection method hitherto commonly employed.

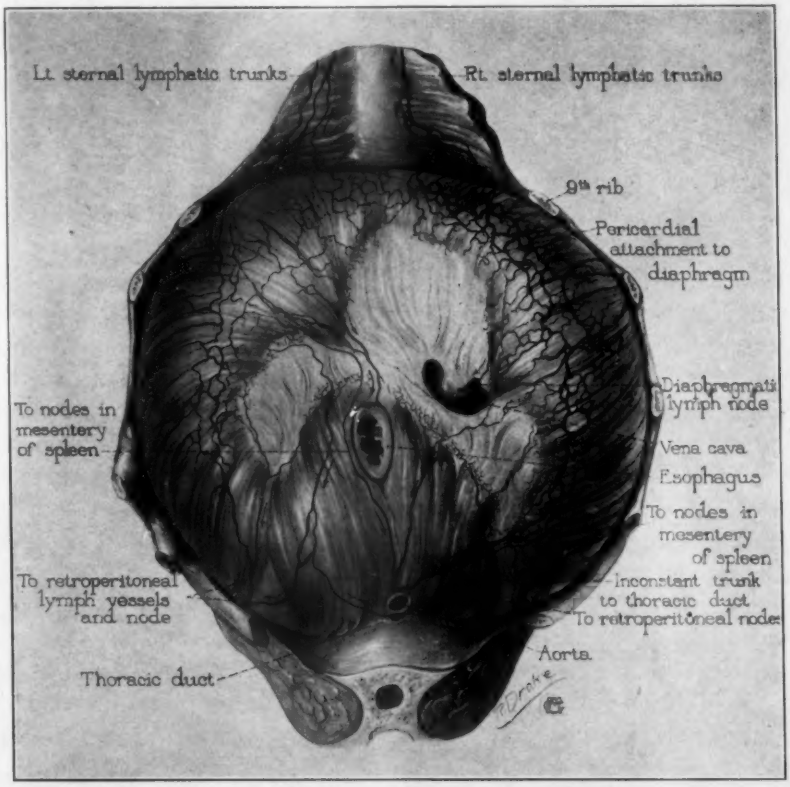

Fig. 1.—The pleural surface of the diaphragm in a dog after the intraperitoneal injection of the graphite preparations. The lymphatic channels of the diaphragm are distended.

Figure 1 shows that the distribution of the lymphatic channels is much greater on the right hemidiaphragm than on the left. The reason for this is not clear, although the proximity of the large right hepatic lobe, providing greater contact surface during respiration, may be a factor. Prolonged massage of the left side of the chest and abdominal wall, however, failed to reveal more lymphatic channels on the left hemidiaphragm than could be seen during the normal process of absorption. The main collecting trunks of the diaphragm lie below the pleura,

on the muscle bundles, and pursue in general a diagonal course from the crura to the para sternalis. These larger channels, restricted for the most part to the medial portion of the muscular area, form a rather prominent plexus as they continue toward the sternum, and the smaller lymphatic vessels which arise along the costal attachment pursue more or less parallel courses and empty into the major plexus. The central tendon is relatively devoid of lymphatic channels. Smaller capillaries, however, are frequently discerned along the margins of the tendons, whence they empty into the larger vessels of the muscular portions and occasionally small channels drain across the central tendon from one muscular portion to another, particularly if the flow is toward the thoracic duct.

There are five drainage routes by means of which the absorbed material is removed from the diaphragm. The first of these is the sternal route, by far the more effective, which receives the drainage from the pars sternalis, pars costalis and to a certain extent from the pars lumbalis. From the standpoint of size and distribution the four remaining routes are apparently of equal significance in lymphatic drainage. The first of these, and perhaps physiologically and pathologically the most significant, comprise a variable group of small lymphatic capillaries which arise, partly in the pars sternalis, just ventral to the central tendon and partly from the muscular portion dorsal to the tendon. This group of small lymphatics, generally more conspicuous on the left side, leaves the diaphragm, courses forward along the pericardium, often parallel to the esophagus and the vagus, to the bronchial lymph nodes which lie dorsal to the base of the lung (fig. 2). Further drainage from this portion of the diaphragm is effected by a small lymphatic channel which usually arises in a small node and courses forward along the phrenic artery and nerve to a lymph node in the anterior mediastinum (fig. 2). The third route, effecting the anterior drainage of the lymph from the diaphragm is by way of the thoracic duct. The dorsal portion of the pars costalis and a portion of the pars lumbalis are drained by a varying number of small lymph channels which empty directly into the thoracic duct. The relative quantity of lymph which pours into the thoracic duct from the diaphragm is exceedingly small; in numerous experiments following the intrathoracic cannulation of the thoracic duct in animals which had received the peritoneal injections of graphite, free particles were rarely encountered in the thoracic lymph. In a single case, rapid appearance of the graphite in the thoracic duct suggested an unusual distribution of the diaphragmatic channels, and subsequent necropsy revealed an exceedingly large channel emptying directly into the thoracic duct. The thoracic duct is a variable structure in the different dogs. It is often double, somewhat

plexiform and passes forward usually to the right of the aorta and ventral to the azygos vein.

The other two lymphatic routes, draining a portion of the diaphragm, lead into the peritoneal cavity or into the retroperitoneal spaces dorsal to the two diaphragmatic crura. Of these two, the latter course is the more extensive and the more constant in its distribution. In figure 1 lymphatic channels are shown in the lower right and lower left margins of the diaphragm, which carry a portion of the lymph backward, dorsal to the peritoneum, to a large lymph node which lies in the retroperitoneal space just above the kidney; thence different channels pass obliquely to the cisterna. In figure 2 a group of three or four lymphatic channels are shown along the dorsal portion of the wall of the chest, coursing relatively parallel to the aorta. These channels, which readily distend with graphite when it is introduced into the peritoneal cavity, join those coming from the adjacent portion of the diaphragm and together course posteriorly to the retroperitoneal node described. Figure 1 shows also a group of three lymphatic channels which course over the dorsal half of the diaphragm and converge near the ventral margin of the aorta. These channels then course directly through the diaphragm, enter the peritoneal cavity, and may be traced directly to a lymph node in the mesentery of the spleen. Invariably these mesenteric nodes are black when the injection residue is washed from the peritoneum, but the extent of the vessels coursing from the diaphragm into the peritoneal cavity is a variable one. In certain dogs we were able to recognize them with great difficulty, and in others such peritoneal channels coursing directly from the diaphragm could be traced directly to the lymph nodes near the spleen, the kidney, and in some instances to the pancreas of Aselli.

We shall summarize briefly the lymphatic drainage from the diaphragm of dogs previously given injections of the graphite preparation. There are five routes through which such particulate matter is removed: the sternal, the pulmonary, the thoracic duct, the retroperitoneal and the direct peritoneal routes. Of these, the sternal route is essentially the most conspicuous and probably carries away four-fifths or more of the material removed from the peritoneal cavity. The pulmonary route is perhaps the next efficient, followed by the thoracic duct, the retroperitoneal lymph ducts, and, lastly, the direct peritoneal course, if one is to judge by relative distensibility following peritoneal absorption.

In the dog, then, the major drainage from the diaphragm is through the sternal lymphatics. In the slightly reflected portion of the sternum, as shown in the upper portion of figure 1, a group of three or four channels course from the diaphragm into the spaces between the intercostal muscles and continue forward, parallel to the sternal or thoracic

artery and vein. Occasionally, plexuses are formed by these channels around the blood vessels. These sternal lymphatic trunks continue into a series of two to four lymph nodes which lie in the interval between

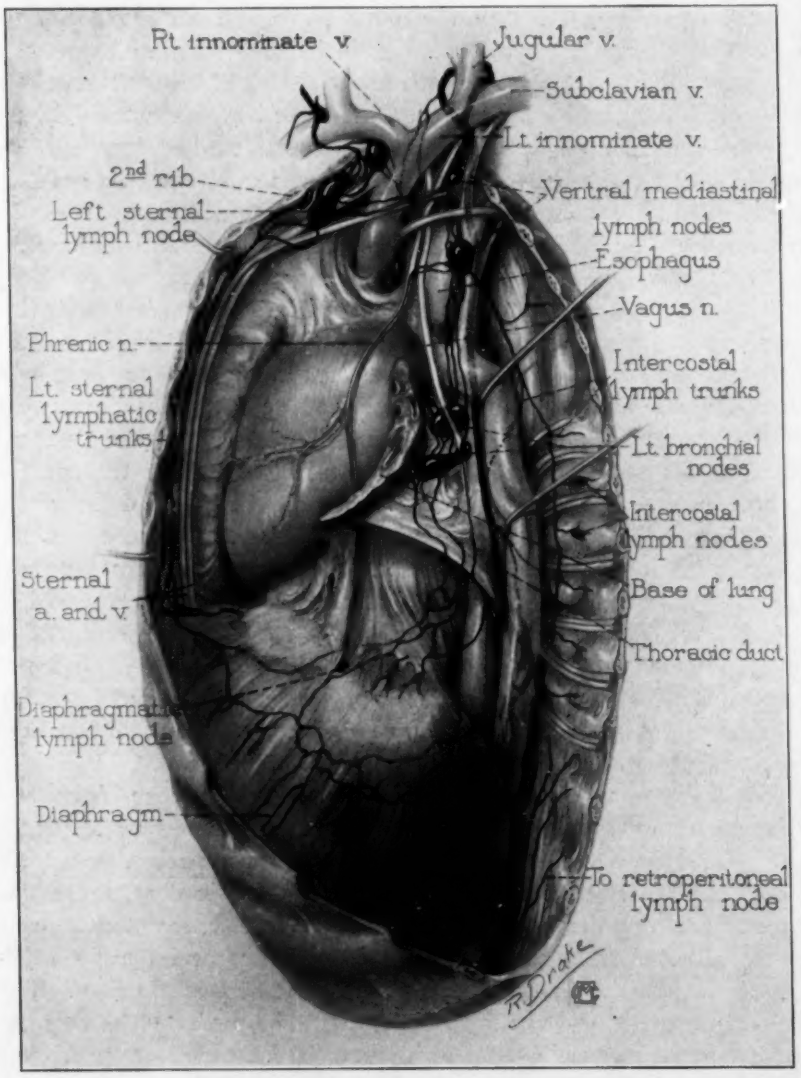

Fig. 2.—The right half of the thorax removed from a dog after intraperitoneal injection of the graphite. The lymph channels and nodes are shown, together with their relation to the thoracic duct.

the first and third ribs (fig. 2). Throughout their forward course the lymphatics on the two sides of the sternum are independent of each other, but in the region of these sternal lymph nodes numerous collecting

branches unite the two (fig. 3). Thence, the lymphatic drainage may be in any one of three directions: directly through collecting channels which course through the fascia along the right innominate vein to the right lymphatic duct and enter the venous system at the right jugular subclavian confluence; through anastomosing channels and additional nodes directly to the thoracic duct near its venous confluence, or by way

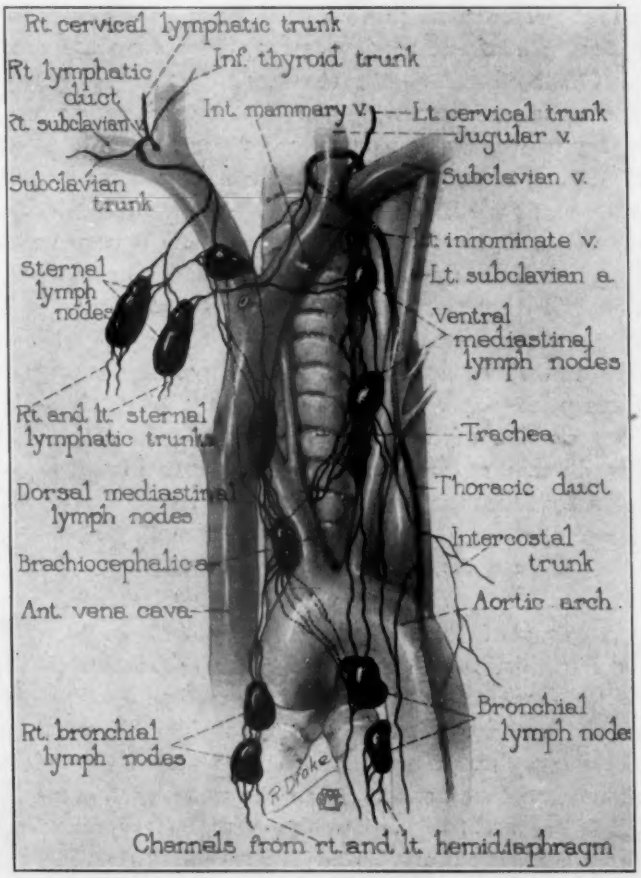

Fig. 3.—Dorsal view of the mediastinal region in a dog, showing lymph nodes and associated lymph channels in a dog given an intraperitoneal injection of the graphite preparation.

of a paratracheal mediastinal lymph node (fig. 3). The pattern of the lymphatic distribution in this region is a variable one, and an attempt will not be made to give in detail the various courses which our dissections have revealed. In general, the drainage from the right sternal lymphatics is to the right lymphatic duct; drainage from the left sternal lymphatics, with the exception of certain anastomoses, is toward the thoracic duct.

Figure 2 shows the drainage from the left side of the diaphragm through the pulmonary region. On the right side, the lymphatic channels from the diaphragm to the hilum of the lung are far less conspicuous, and yet in nearly every animal studied one or more such vessels were seen leading directly to parabronchial nodes on the right side. The lymph duct which courses forward over the pericardium, parallel to the phrenic nerve is invariably present. This empties directly into a paratracheal mediastinal lymph node, lying directly on the ventral surface of the trachea, somewhat anterior to the arch of the aorta. Other channels leave the diaphragm in the region of the central tendon, incline somewhat dorsally and course either through the pulmonary mesentery or along the esophagus parallel to the vagus nerve. This group of lymphatics enters the parabronchial nodes, usually two or three in number, which lie within the arch of the aorta just dorsal to the base of the left lung (fig. 2). We have never observed any direct lymphatic continuity between these diaphragmatic channels and the tissue of the lung. The pulmonary lymph channels, however, communicate directly with these parabronchial nodes and accordingly any retrograde flow of lymph would permit dispersion of the particulate matter of the peritoneal cavity within the lung. The discoloration of the lung in a number of dogs would suggest such a reversal, and yet the varying degrees of anthracosis which occur in the average dog render such a conclusion untenable.

Anterior to the parabronchial nodes, the lymph drainage continues by way of channels which pursue rather different courses in different animals. The more usual pattern encountered is that illustrated in figure 3. A group of from five to eight channels continues forward from these bronchial lymph nodes to a series of mediastinal nodes which lie in the fascia along each side of the trachea, the brachiocephalic artery and the anterior vena cava. A considerable portion of the lymph from the nodes on the left bronchus passes through channels which lie dorsal to the aortic arch, to the more posterior mediastinal node, which lies adjacent to the brachiocephalic artery, and receives the drainage from the right bronchial nodes as well. Other channels from the left bronchial nodes course directly forward ventral to the aortic arch and empty into the paratracheal node at a considerable distance anteriorly. Figure 3 shows that the drainage from the right side of the pulmonary region, and no doubt to a considerable extent from the left, empties into the venous circulation through the right lymphatic duct. The principal drainage from the left side, however, although exhibiting certain anastomotic arrangements with that of the right, is more largely forward through a series of two or three nodes which lie along the left side of the trachea and which ultimately connect by additional channels with the thoracic duct near its venous confluence.

The third anterior drainage route from the diaphragm is by way of the thoracic duct. As shown in figures 1 and 2, only a few small lymphatic channels ordinarily pass from the diaphragm into the thoracic duct. Accordingly, this route is of relatively small significance in the drainage of particulate matter from the peritoneal cavity. The duct, somewhat plexiform in arrangement, courses forward to the right of the aorta and ventral to the azygos vein, much as is shown in standard comparative textbooks, forms the characteristic ring around the base of the jugular vein and empties into the vascular circulation either at the subclavian confluence or into the left innominate or left brachiocephalic vein.

SUMMARY AND CONCLUSIONS

From this rather cursory survey of the anatomic relations of the lymphatic channels which function in the removal of particulate matter from the peritoneal cavity of the dog, it is evident that the thoracic duct plays a relatively insignificant part. Particulate graphite, when injected directly into the peritoneal cavity, is readily visible in the diaphragmatic lymphatics and the sternal trunks within from ten to twelve minutes in normal healthy dogs. In animals, however, in which the respiratory mechanism is disturbed, so that the normal excursion of the diaphragm is modified somewhat, the interval between the peritoneal injection and the appearance of the graphite in the sternal lymphatics is greatly prolonged. The injected graphite is visible in the sternal lymphatic trunks usually long before it can be identified in the cannulated thoracic duct. In only one dog of our series, a more or less rapid appearance of the pigment in the thoracic duct was explained at necropsy when an unusual distribution of the lymphatics of the dorsal portion of the diaphragm was discovered to include major channels which were directly confluent with the thoracic duct. Ordinarily, the lymph of the thoracic duct is colored only lightly and then only after prolonged intervals, when the lymphocytes within it contain large numbers of the graphite particles, probably phagocytes within the peritoneal cavity. With the animal under ether anesthesia, it is relatively simple to open the median line of the chest in the region of the first or second costal cartilages and thus expose the sternal or thoracic blood vessels. In this way the region may be watched and the first appearance of the black graphite in the clear lymph of the channel may be noted. Preparations made of the lymph coming through these channels show that the graphite contained within it is in the free particulate state, just as it was injected into the peritoneum. On the other hand, smears made from the lymph nodes in this region, as well as fixed paraffin sections, show that the material is both in the cells and in the free state, the particles having been phagocytosed by the large cells of the lymph node.

If in the same animal, or in one subjected to the same injection into the peritoneum, a cannula is introduced into the thoracic duct in the neck near its venous confluence, samples of lymph may be taken for analysis. Within thirty minutes, following the peritoneal injection, darkly colored lymph may be recovered from the thoracic duct in the neck. Analysis shows that the graphite contained therein is partly free and partly in the large lymphocytes. The cells containing the graphite are probably derived from the lymph nodes of the pulmonary, the tracheal or the sternal regions; the particles in the free state have passed directly into the lymph stream from the diaphragm.

By means of insufflation, one of us (Graham) was able to develop a technic whereby the thoracic portion of the thoracic duct could be cannulated and samples of lymph could easily be collected for analysis of cellular content. In a number of experiments, lymph was collected from the thoracic duct of dogs that had previously received a peritoneal injection of the graphite preparation. The sample taken immediately after the intraperitoneal injection was normal lymph with the usual number of small and large lymphocytes. During the first hour, long after the sternal lymphatics were black, there was no evidence of the graphite either in the free state or in phagocytic cells, yet there was an apparent increase in the number of large lymphocytes. An hour and a half following the injection, the larger cells of the lymph contained many granules of graphite, but the smaller cells were entirely devoid of them. There were no free particles in the lymph stream at this time. Subsequently, with an increase in the number of these large cells, small quantities of free graphite appeared in the lymph. Also, at intervals of about two hours following the injection, the larger cells contained the graphite in varying quantities, and in many cases the cytoplasmic bodies were literally packed with granules. Analysis of the peritoneal exudate showed masses of cells, similar to those recovered from the thoracic duct, packed with the graphite material. Unquestionably, these cells of the thoracic duct are identical with those in the peritoneal exudate, and they had probably entered the cisterna after phagocytosing the graphite in the peritoneal cavity. The particles of graphite which were encountered free in the lymph of the thoracic duct probably entered the duct through the few small channels coming from the dorsal margins of the diaphragm. Samples of blood taken from the femoral artery at this time contained occasional granules of free graphite, together with cells moderately packed with the injected material.

If one is to judge by the degree of physiologic activity and the number of lymph channels leading from the diaphragm it is apparent that the lymphatic paths coursing through the pulmonary region are more effective in removing foreign particles from the diaphragm than the thoracic duct. These channels are not large and by no means com-

parable to those which run along the sternum, and yet they are invariably present on the left side, coursing forward through the related mesenteries to the lymph nodes at the base of the lung, and usually are identified on the right side, although perhaps to a less degree.

In the removal of foreign particulate matter or bacteria from the peritoneal cavity, these pulmonary lymphatic routes are perhaps the most significant from the standpoint of pathology. Although these channels are not directly confluent with pulmonary lymph vessels yet they join with them in the nodes which lie at the base of the lung. In these nodes the lymph draining the two regions mingles and courses forward in the channels of the mediastinum. Accordingly, although direct pulmonary contacts with peritoneal drainage is not effected, nevertheless any disturbance in the flow or lymph coming from the lung, so as to involve stasis or even a retrograde flow, could well infect the lung with peritoneal organisms. These observations do not warrant such conclusions, for the pigmented lung so often encountered in these experimental animals has, of course, other explanations. The common pulmonary complications which accompany abdominal operations on dogs, however, may have their explanation in the lymphatic association of the two regions.

OLD NODULAR GOITER SURROUNDING TRACHEA, POSTERIOR TO THE CAROTID WITH THE ISTHMUS POSTERIOR TO THE ESOPHAGUS

REPORT OF A CASE WITH SUDDEN DEATH FROM ACUTE ABSCESS *

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FORT WAYNE, IND.

History.—S., a man, aged 60, was brought to the hospital suffering from retention of urine due to an enlarged prostate. He had a large, nodular, hard goiter which was fixed and which he said never bothered him. However, on inquiry, it was found that for six or eight months he had been unable to breathe well when lying flat, and that for the same length of time his voice had been affected. There were no toxic symptoms. He had had the goiter "ever since he could remember."

Catheterization, with the usual treatment after ten days or two weeks, put the patient in a condition for operation. A two-stage prostatectomy was done on December 24 and January 15, respectively. The patient was making an ideal recovery until January 21, when he had a chill accompanied by severe pain in the right side of the goiter and a temperature of 103.4 F. He also complained of difficulty in breathing. On the next morning when I saw him, he was sitting in bed reading the paper, and complained only of some soreness in the right side of the goiter. The temperature dropped to normal that night, and on the 23d he was feeling quite well, with a slight elevation of temperature and some soreness still present in the goiter. Early the next morning (the 24th), he complained of severe pain in the neck and difficult breathing. Within ten minutes he was dead.

The nurse in charge of the floor also reported that the goiter was greatly increased in size, and that the man was cyanotic and evidently died from asphyxia. Both drains were removed from the bladder on the 23d when the cystotomy wound seemed to be in a good condition, and the drainage was satisfactory.

Autopsy.—Autopsy was performed three hours and thirty minutes after death. The bladder, the area around the prostate and the cystotomy wound were clean and normal. The abdomen was normal, except for stones in the gallbladder. Extensive pleural adhesions (old) were found on the left, and there were some on the right, also. The left ventricle was slightly hypertrophic. The cardiac vessels and valves were normal. No thrombus or embolus could be found.

The large goiter was uncovered with difficulty on account of close old adhesions; during the process of uncovering it, an abscess on the right side was opened and discharged more than an ounce of sanious pus, which was found to be streptococcic. Sharp dissection was necessary in removing the gland, which was closely adherent throughout its entire surface.

The right lobe (the larger) was crossed vertically on the outer anterior aspect by a white, flat band, which proved to be the carotid artery (fig. 1). This was separated from the gland, and the latter was removed in its entirety. The right lobe crossed over the trachea and was intimately adherent to the front of the

* Submitted for publication, April 2, 1929.

left lobe, and the isthmus was behind the esophagus. Thus, the esophagus and trachea were completely encircled by the goiter (fig. 2).

Figure 1 is a drawing that I made from memory immediately after the removal of the gland. Neither the artery, the trachea nor the esophagus was removed with the goiter but, to make the interpretation clearer, the drawing was made as though they had been. The artery should appear more flattened than it does in the drawing.

Figure 2 is a schematic representation of a cross-section showing the relation of the artery, the trachea and the esophagus to the goiter, also the location of the isthmus. Microscopic examination of several sections showed no malignant disease but old and recent inflammatory reactions.

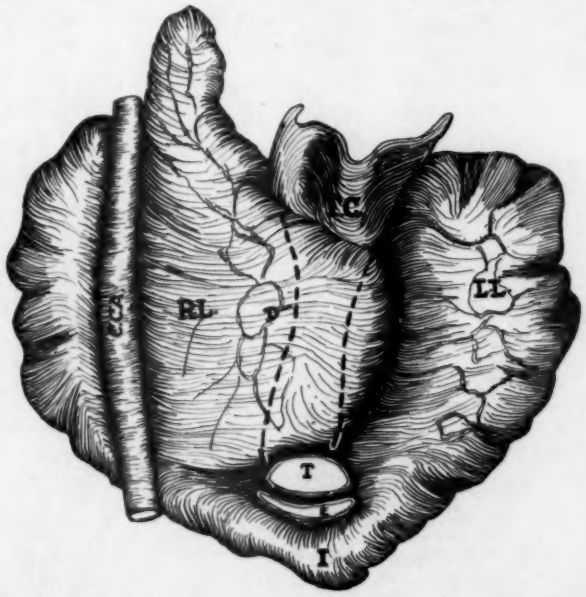

Fig. 1.—Drawn immediately after removal, from memory. *C.C.A.*, flattened carotid; *D*, dent in trachea made by pressure of goiter; *T.C.*, thyroid cartilage; *T*, trachea; *E*, esophagus; *I*, isthmus; *R.L.* and *L.L.*, right and left lobes, respectively.

COMMENT

In my opinion, the sudden death in this case was due in the main to an acute streptococcic abscess. The source of the infection was not determined.

The universal close adhesions prove the existence of a long-standing, low-grade inflammation, an exacerbation of which may have been caused by the lowered resistance consequent on the urinary trouble, plus the operation. It should be added that examination of the specimen after removal showed several additional abscesses.

The asphyxia may have been in part, at least, due to pressure on the superior laryngeal nerve, as the abscess was located in the upper right aspect of the gland. However, the dent remaining in the trachea after the gland was removed shows that the capacity of that tube had been encroached on for a long time.

From the flattening of the carotid, one would suppose that the passage of blood through this vessel was considerably inhibited. If so, there were no clinical signs indicative of it.

I never saw the isthmus located behind the esophagus before, nor have I found a similar case reported, although, as is well known, unusual forms of this part of the thyroid are not infrequent. Absence of the isthmus or of one of the lobes is not extremely uncommon; it is quite possible that in the case reported, the isthmus and the left lobe were never developed and that what seemed to be the isthmus was really an extension of the right lobe of the gland, as was what I have named the left lobe.

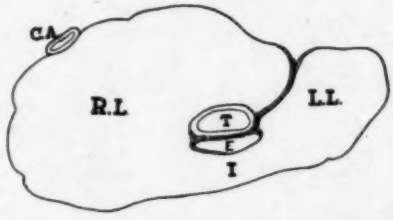

Fig. 2.—Schematic drawing of cross-section, showing relation of artery (*C.A.*), esophagus (*E*) and trachea (*T*) to the goiter. *L.L.*, left lobe; *R.L.*, right lobe; *I*, isthmus.

Pemberton [1] reported a case (fig. 3) in which there was a mass of thyroid tissue behind the trachea and esophagus which had developed from the right lobe and had formed a tumor on the left side from which the lobe had previously been removed. Hertzler sent me a photograph of a case much like Pemberton's (fig. 4), except that the growth was malignant.

Graham [2] referred to a case reported from Hoffman's Clinic by M. Willenbacher, in which the goiter completely encircled the trachea, producing dyspnea in a new-born child, and which was successfully removed forty-three hours after birth.

The distortion of the great vessels that was present in the case here reported has been seen in "several instances" in the Lahey Clinic [3] in

1. Pemberton: Surg. Clin. N. Amer. **6**:1278 (Oct.) 1926.

2. Graham: Practical Medicine Series, Surgery, Chicago Year Book, 1928, p. 211.

3. Clute, H. M., and Smith, L. W.: Cancer of the Thyroid Gland, Arch. Surg. **18**:part 1, 14 (Jan.) 1929.

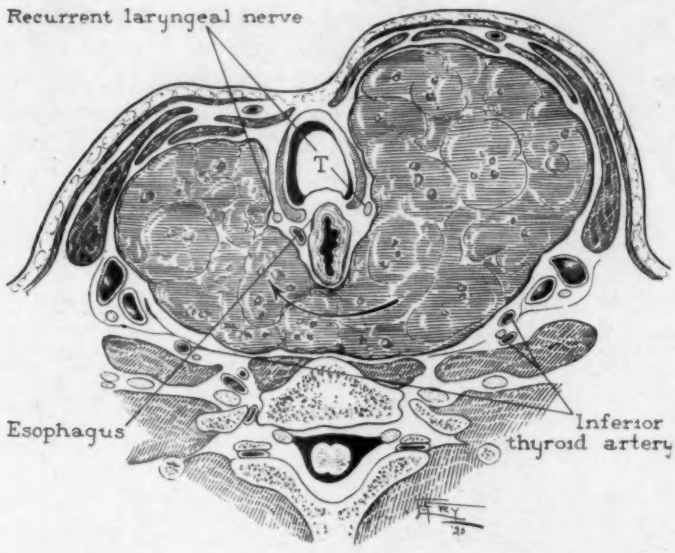

Fig. 3.—Cross-section of a huge retrotracheal projection, forming a tumor on the left side of the trachea from which the lobe had been previously removed. (From Pemberton: Surg. Clin. N. Amer. **6**:1278 [Oct.] 1926.)

Fig. 4.—Postmortem specimen from Hertzen's case of sarcoma.

patients with cancer of the thyroid. Lahey [4] said that they have never had a case like the one here reported. Albert Kocher [5] referred to an outward displacement of the vessels so that the "carotid is felt in the external posterior portion of the tumor underneath the skin and along the outer border of the sternocleidomastoid muscle," but made no mention of such distortion as occurred in this case.

Crotti [6] spoke of the displacement of the vessels much as Kocher did, but made no mention of anterior displacement.

Neither Ochsner [7] nor Crile,[8] in their works on goiter, mentioned derangement of the vascular relations such as is herewith reported.

C. H. Mayo [9] said that he had seen "a few cases, not more than four" like the one reported here.

Crotti,[10] said that he had seen eight or ten cases of "circular goiter in which the two lobes came in contact behind the esophagus."

SUMMARY

A case is reported of sudden death from asphyxia due to an acute streptococcic abscess engrafted on a chronic inflammation in an old, nodular, nonmalignant goiter, which completely surrounded the trachea and esophagus with the isthmus behind and the lateral lobes meeting in front of the trachea, and having the carotid vessels stretched over the front of the right lobe. It is probable that both the anterior dislocation of the vessels and the retrotracheal projection of the gland are more likely to occur in malignant goiters.

Cases similar in some respects are referred to, but after considerable inquiry by letter and research in the literature, no case like the one here reported has been found.

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4. Lahey: Personal communication to the author.

5. Kocher, Albert, in Keene: Surgery, Philadelphia, W. B. Saunders Company, 1908, vol. 3, p. 359.

6. Crotti, Andre: Thyroid and Thymus, Philadelphia, Lea & Febiger, 1918, p. 124.

7. Ochsner and Thompson: Thyroid and Parathyroid Glands, St. Louis, C. V. Mosby Company, 1910.

8. Crile, G. W., and his associates: The Thyroid Gland, Philadelphia, W. B. Saunders & Co., 1922.

9. Mayo, C. H.: Personal communication to the author.

10. Crotti: Personal communication to the author.

THE EFFECT OF INTRABRONCHIAL INJECTIONS OF IODIZED POPPY SEED OIL 40 PER CENT

AN EXPERIMENTAL STUDY ON DOGS *

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When iodized poppy seed oil 40 per cent was first advocated in intrabronchial instillation for the purpose of lung mapping, there seemed to us to be several theoretical objections. One of these was that a fibrosis of the lungs would be produced as a result of prolonged presence of an intrabronchial foreign body (iodized oil). Another was that bronchitis or bronchopneumonia would develop due to the transplantation of bacteria from the pharynx into the finer bronchioles by means of the oil. Long before we found experimentally that this second objection was theoretical only, we were convinced by reports of the clinics in which iodized oil 40 per cent had been taken up enthusiastically from the start that bronchitis and pneumonia did not follow its use.

The validity of the first objection, however, seemed to us to be strengthened as we began to see patients a year or two after the injection of iodized oil in whom traces of the oil remained visible roentgenographically. Also, Pinkerton [1] reported several cases of children in whose lungs marked fibrosis had occurred following the aspiration of small quantities of petrolatum which had been instilled into the nose for some local nasal condition. Finally, in our own hospital, Dr. Irving Stein found marked fibrosis of a fallopian tube which contained iodized oil several months after the oil had been instilled into the uterus for the purpose of mapping the uterine cavity and tubes.

EXPERIMENT

Our experiment consisted of intrabronchial injection of a small quantity of iodized poppy seed oil 40 per cent into dogs; the animals

* Submitted for publication, Jan. 15, 1929.

* From the Nelson Morris Institute for Medical Research of the Michael Reese Hospital, and from the Department of Experimental Surgery, University of Illinois.

1. Pinkerton, H.: Oils and Fats; Their Entrance Into and Fat in Lungs of Infants and Children, Am. J. Dis. Child. **33**:284 (Feb.) 1927.

were then killed at various intervals from a few days to seven months after the injection, and the lungs were studied roentgenographically, macroscopically and microscopically.

The technic carried out was identical in all dogs.

The animals were given hypodermic injections of morphine of sufficient strength to make them drowsy and docile, but not enough to put them to sleep.

They were placed on a fluoroscopic table and strapped to a board. Their mouths were held open by an assistant, and a tube (soft rubber French catheter no. 12)

Fig. 1.—Roentgenogram taken immediately after the injection of iodized oil into the lower branch of the right bronchus.

was quickly slipped through the posterior pharynx into the trachea and fed downward until its tip was in one or the other of the two main bronchi. Then from 5 to 20 cc. of iodized oil was injected into the bronchus under fluoroscopic control. The amount varied according to the size of the dog. Sufficient iodized oil was injected to outline clearly the bronchus and bronchioles of one or two lobes of a lung. The catheter was withdrawn and a roentgenogram immediately made.

The dogs were allowed the liberty of the kennels. At frequent intervals, roentgenograms were made.

The dogs were killed at lengths of time varying from a few days to seven months after the injection of iodized oil. A careful and complete autopsy was

done on each dog. The lungs were removed in toto, and roentgenograms were made before they were examined and sectioned.

Sections were made from every lobe of each lung and were stained with hematoxylin and eosin and sudan III (fat stain).

The control consisted of dogs killed for other experimental work. The lungs were removed in toto; roentgenograms were made, and the lungs were sectioned as in the experimental dogs.

RESULTS

Only two of the twelve dogs developed pneumonia—one dog a few days after injection and one several months after. We feel that these

Fig. 2.—Picture of same dog in figure 1 taken four months later, showing retention of the iodized oil into the lower part of the right lung, and also the retention of the same material in the upper lobes.

two deaths have no bearing on the experiment, as in all our dogs the mortality from pneumonia during the winter of 1926-1927 was greater than this; furthermore, one of these dogs died in an epidemic which killed nearly all the dogs in the kennels at that time.

The fact that ten of twelve dogs did not develop pneumonia would have led us to discard the theoretical objection that iodized oil might cause pneumonia, if the clinical evidence had not already caused us

to do so. Macroscopically, only one dog showed signs of marked pulmonary fibrosis and scarring. This was in a dog in which the iodized oil had been injected one month previously. The appearance of the lung suggested a much older process, and the fact that we have seen dogs into which injections had never been made and which had similar lesions leads us to the conclusion that the fibrotic changes in this case cannot be traced to the iodized oil.

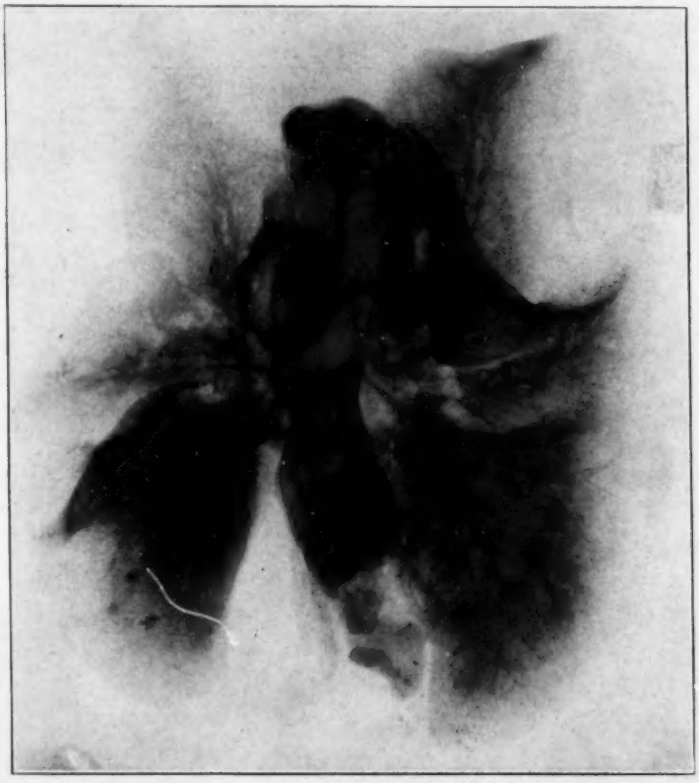

Fig. 3.—Roentgenogram of lung removed after the dog was killed six months after injection, showing retention of iodized oil.

In almost every instance, the oil did not remain in the lobes injected but was quickly dispersed. In every instance almost all of the oil had disappeared by the time the second roentgenogram was taken.

In every case, small traces of iodized oil were seen roentgenographically in the lungs removed at autopsy. That includes the dog killed after almost seven months.

The results of the microscopic examinations were as follows:

The lungs of the dogs into the bronchial tree of which iodized oil had previously been injected did not differ grossly in any way from the

lungs of control animals. Microscopically, slight changes were present in the dogs given iodized oil. These changes were not constant in all the sections and did not vary in degree in any single section. Sections taken from those areas which showed a slight degree of mottling in the roentgenogram taken of the bare lungs after autopsy did not present signs or changes any more unusual than those in areas which were clear in the roentgenograms.

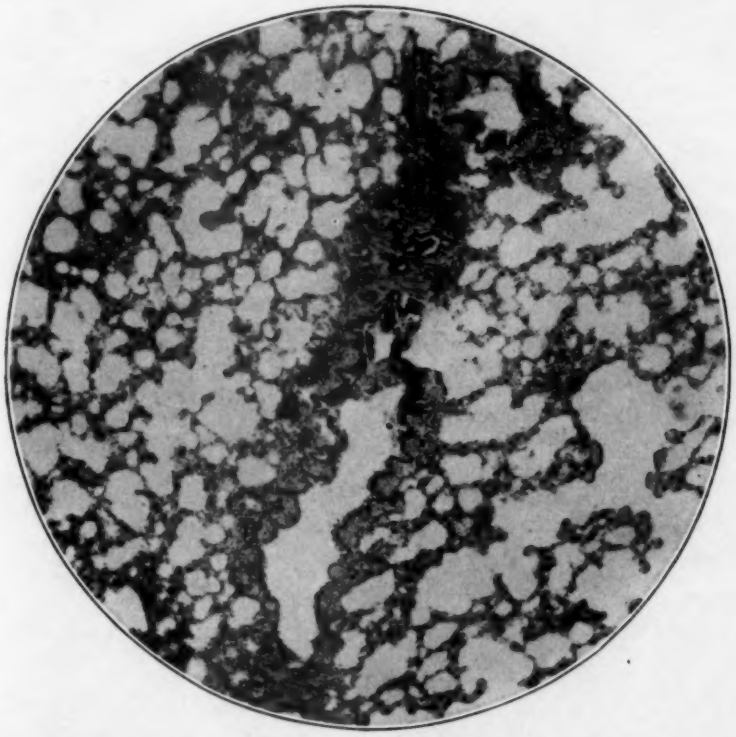

Fig. 4.—The portion of the lung which on roentgen examination was shown not to contain iodized oil. It is impossible to distinguish this section from sections of the control animals.

In sections stained with hematoxylin and eosin in both the dogs given iodized oil and in the control animals, a moderate number of large mononuclear phagocytes containing small particles of pigment occurred beneath the pleura. The number and distribution of these cells were about the same in the two sets of dogs. These cells often occurred in the region of small anthracotic areas, or occurred singly.

In the animals given iodized oil the terminal bronchioles were slightly more prominent than those of the controls, and there occurred a slight

increase in the thickness of the wall and a not prominent infolding of the epithelium. The number of lymphocytes in the walls of the bronchi was about the same in the two sets of animals.

In the dogs given iodized oil, small focal areas of fibrosis occurred occasionally beneath the pleura and about the terminal bronchi, and while similar areas occurred in the control animals, their frequency and size were slightly more prominent in the dogs given iodized oil.

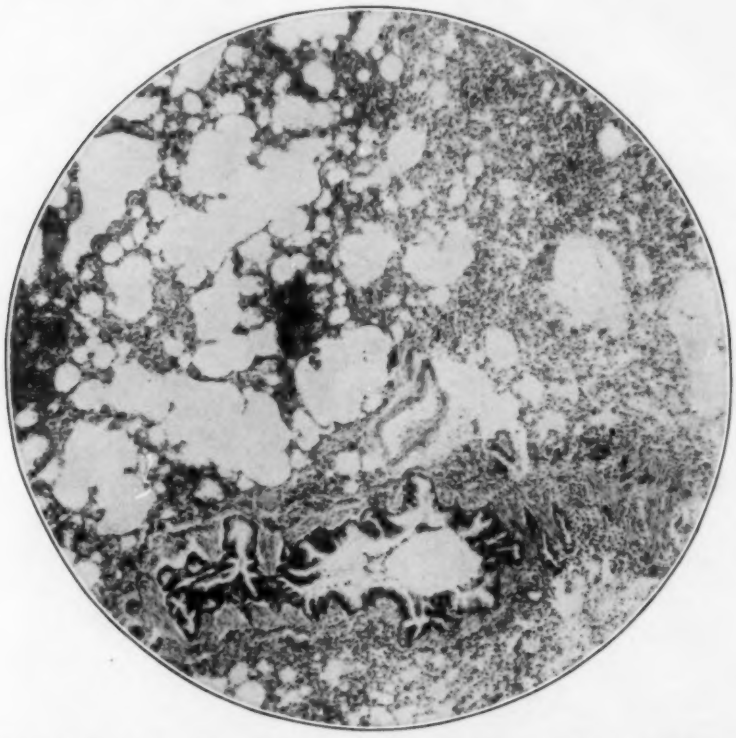

Fig. 5.—The lower lobe of the right lung, showing a slight thickening of the parenchyma. In the control dogs, apparently normal lungs showed areas indistinguishable from these.

In sections stained with sudan III, the number of macrophages containing small refractile globules of fat were slightly increased in the dogs given iodized oil. These macrophages occurred beneath the pleura, usually about a small area of anthracosis, but frequently appeared singly. An occasional lining alveolar cell containing small fat globules was present.

The larger bronchi of the dogs given iodized oil infrequently showed fat within the wall. This small amount of fat was located within the fat cells.

The conditions just described were not prominent in any of the sections taken from the lungs of dogs given iodized oil. They were present to a slightly less degree in the lungs of control animals.

Our results have led us to drop our theoretical objections to the use of iodized oil. Our results correspond with clinical experience and also with experimental observations published by others.

We were keenly interested in the result of Pinkerton's [2] experimental work. He showed results almost identical with our own when iodized oil was used, and results similar to those found in the lungs of children when mineral oils were used. He advanced the explanation that the difference in the reaction might be explained on the basis of the presence of specific ferments-lipases. At the suggestion of Dr. Schultz, the director of the Nelson Morris Institute, we attempted to show the presence of these ferments in lung tissue. However, the usual methods did not give results, showing that if these ferments do exist in lung tissue they exist in small quantities, perhaps in quantities not greater than those found in the circulating blood.

SUMMARY

Instillation of iodized poppy seed oil 40 per cent into the bronchial tree of dogs is not followed by pneumonia nor, as far as we could see, by any acute cellular reaction.

Most of the iodized oil is speedily expelled by coughing.

Small amounts of the oil are retained for at least seven months.

The presence of this iodized oil apparently does not cause a definite foreign body reaction.

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2. Pinkerton, H.: Reaction to Oils and Fats in Lungs, Arch. Path. **5**:380 (March) 1928.

INTESTINAL OBSTRUCTION

EXPERIMENTAL STUDIES ON TOXICITY, INTRA-INTESTINAL PRESSURE AND CHLORIDE THERAPY *

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In spite of the large amount of experimental and clinical work done on intestinal obstruction, there are still numerous insular concepts of the cause of death, and there are differences of opinion as to treatment. There have been several excellent reviews of the literature during the last few years, and Cooper has recently published an admirable analysis of the situation from the experimental evidence at hand. Since this literature is available, we shall not attempt to review past evidence but shall report the results of our experiments and our deductions as to the manner of treatment likely to be most beneficial for patients having intestinal obstruction.

There is general agreement that simple obstruction differs materially from obstruction with strangulation. This study deals only with simple obstruction.

There have been three primary purposes in these experiments: (1) to determine the toxicity of the content of obstructed intestine when it is present in obstructed and nonobstructed bowel; (2) to determine the effects of varying intra-intestinal pressure on the length of life after obstruction is induced, and (3) to determine the therapeutic value of sodium chloride under varying conditions of intra-intestinal pressure.

The attempt to answer these questions has necessitated inquiry into several subsidiary issues which will be discussed as they appear.

Rabbits have been used as the experimental animals. They are not so satisfactory to use as dogs, for they are more delicate and live a shorter period after obstruction. This disadvantage is more than offset in these particular experiments by the fact that rabbits do not vomit and therefore their intra-intestinal pressure can be controlled.

TECHNIC OF PROCEDURES

All operations, except the second stage of some jejunostomies, were done with the animals under ethylene anesthesia with no preliminary medication. The animals were denied food for forty-eight hours but were allowed water preoperatively. Operations were performed under aseptic precautions, and virtually no wound infections developed.

* Submitted for publication, March 30, 1929.
* From the Laboratories of Columbia Hospital.

Obstructions were created in two ways. Temporary obstruction was produced by making an aperture in the mesentery just beneath the bowel and passing a one-fourth inch rubber band through the opening. The ends of the rubber band were brought out through stab wounds in the ventral abdominal wall about three eighths of an inch apart. The ends of the band were then sewed together applying sufficient tension to compress the intestine so as to occlude the lumen but not enough to destroy the blood supply. This tension was applied with the intestine under observation. When the obstruction was to be released, the rubber band was cut and removed without anesthesia. Usually the intestinal contents could immediately be heard passing beyond the point of obstruction. Permanent obstruction was produced by tying umbilical cord tape around the bowel.

Jejunostomies were performed to permit complete drainage of the stomach and duodenum. Although three methods were tried, none was entirely satisfactory. The principal difficulty was in obtaining free drainage with no back pressure as shown by duodenal distention.

The first method consisted of severing the intestine, tying off and inverting the aboral end, placing a small rubber tube in the proximal end and bringing it out through the abdominal wall.

The second method was similar except that no tube was used, and the wall of the intestine was sutured to the skin.

The third method was the most satisfactory and was done in two stages. Under ethylene anesthesia the upper jejunal loop was brought out through the muscles of the abdominal wall and the muscles approximated with the mesentery intervening. The skin was then sutured over the intestine. Several rabbits died of obstruction following this first stage, but some survived and were in good health. The second stage was done under infiltration with procaine hydrochloride. The skin was opened, the intestine severed and the aboral end tied off. Thus there was no soiling of the peritoneum and no change made in the position of the proximal end to interfere with complete drainage.

Gastrostomies were performed in conjunction with several obstructions. After completion of the obstruction, the stomach was sutured to each side of the abdominal incision over a distance of about 2 inches. The stomach was then opened and the mucous membrane sutured to the skin, thus permitting constant emptying of the stomach through the ventral abdominal wall.

Aspiration of stomach content by a catheter through the esophagus was found to be possible only when rabbits had been on an exclusive diet of lettuce, bread and water for from two to three weeks. After forty-eight hours' starvation the stomach was nearly empty, and the secretions accumulating after obstruction could be aspirated through a catheter.

Material to be put into the stomach after the intestine was obstructed was forced through a catheter.

Solutions of dextrose or sodium chloride were injected into the veins of the ear.

Blood for analysis was obtained from the veins of the ear or from the heart.

The nonprotein nitrogen of the blood was determined by the method of Folin and Wu.[1] The blood chlorides were determined by the method of Whitehorn.[2]

Blood for the determination of the alkali reserve was obtained from the heart and oxalated under oil. The method used was that of Van Slyke and Cullen,[3] and Van Slyke.[4]

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1. Folin, D., and Wu, H.: J. Biol. Chem. **38**:81, 1919.
2. Whitehorn, J. C.: J. Biol. Chem. **45**:449, 1921.
3. Van Slyke, D. D., and Cullen, G. E.: J. Biol. Chem. **30**:289, 1917.
4. Van Slyke: J. Biol. Chem. **30**:34, 1917.

The chlorides in the stomach and duodenal contents were determined in the following manner. A weighed portion of the stomach or intestinal contents was made distinctly alkaline with sodium carbonate, the mixture completely ashed, and the residue dissolved in distilled water containing nitric acid. In all, the equivalent of nitric acid was 5 cc. of a 1.42 specific gravity acid. From this point the procedure was the same as that for the determination of chlorides in the blood.

Some rabbits died from the obstruction sooner than was expected so that complete data on their blood were not obtained. Others contracted pneumonia soon after operation. Records of the experiments on such animals were discarded.

THE TOXICITY OF THE CONTENTS OF OBSTRUCTED INTESTINE

Rabbits vary considerably in the length of time they live after obstructions at similar points in their intestinal tracts. The amount of

TABLE 1.—*Changes in Blood Chlorides and Nonprotein Nitrogen Following Intestinal Obstruction at the Levels Indicated*

Rabbit		Before	12 Hr.	14 Hr.	20 Hr.	22 Hr.	24 Hr.	26 Hr.	39 Hr.	43 Hr.	49 Hr.	
3	Nonprotein nitrogen	54.5	..	111.0	..	..	..	..	..	..	..	Middle duodenum
	Chlorides	0.430	..	0.304								
2	Nonprotein nitrogen	82.1	224.5	..	..	..	..	..	..	..	..	Middle duodenum
	Chlorides	0.431	0.355									
10	Nonprotein nitrogen	44.4	..	..	78.9	..	..	..	..	..	..	Proximal jejunum
	Chlorides	0.495	..	..	0.460							
11	Nonprotein nitrogen	43.4	..	..	..	..	133.1	..	..	..	..	Proximal jejunum
	Chlorides	0.502	..	..	..	..	0.409					
18	Nonprotein nitrogen	32.4	..	..	..	..	..	120.0	..	..	..	Proximal jejunum
	Chlorides	0.393	..	..	..	..	..	0.346				
4	Nonprotein nitrogen	44.5	..	..	..	178.5	..	..	267.8	..	..	18 inches jejunum
	Chlorides	0.436	..	..	..	0.399	..	..	0.360			
8	Nonprotein nitrogen	39.5	..	..	..	..	..	..	..	214.3	..	Upper part of ileum
	Chlorides	0.482	..	..	..	..	..	..	..	0.339		
79	Nonprotein nitrogen	65.2	..	..	..	..	104.0					Terminal ileum
	Chlorides	0.475	..	..	..	..	0.420					
9	Nonprotein nitrogen	42.2	..	..	..	..	..	..	..	173.3	178.0	Terminal ileum
	Chlorides	0.455	..	..	..	..	..	..	..	0.419	?	
13	Nonprotein nitrogen	37.0	..	..	..	47.9	..	..	..	..	38.2	Operative manipulation
	Chlorides	0.466	..	..	..	0.461	..	..	..	..	0.471	
28	Nonprotein nitrogen	50.0	..		..	..	47.0	..	..	..	..	Starvation only
	Chlorides	0.507	..	..	..	..	0.498	..	..	..	..	

change in the nonprotein nitrogen and chlorides in the blood also varies somewhat, but as a rule is a dependable indication of the rabbit's condition. Obstruction in the middle of the duodenum is compatible with life for from fourteen to eighteen hours. Obstruction at the beginning of the jejunum caused death in from twenty-two to thirty hours. The duration of survival increases as the obstruction is lower so that life may last from eighty to two hundred hours when the obstruction is at the terminal ileum. These figures hold for rabbits receiving neither food nor water after the obstruction is created. Death occurs more rapidly if food or water is taken.

Contrary to some opinions, it will be noted that obstruction low in the small intestine causes as great a drop in blood chlorides as that fol-

lowing high obstruction, provided there is a proportionate increase in the duration of the obstruction. There is always an increase in the nonprotein nitrogen of the blood as well as in the alkali reserve.

When rabbits are nearing death from obstruction, there is a great decrease in body temperature. Blood pressure is low, and there is some concentration of blood as noted in the diminished amount of plasma in proportion to the cellular constituents. When the rabbits are sick, it is difficult and sometimes impossible to obtain blood from the veins of the ear, even after application of heat and xylol.

The release of an obstruction when the rabbit is nearing death induces a rapid change in its condition. Within an hour its body tem-

TABLE 2.—*Recovery After Release of Temporary Obstruction. The Time Intervals Tabulated Are Those After the Release of Obstruction*

Rabbit		Before	Release	1 Hr.	2 Hr.	3 Hr.	3½ Hr.	4½ Hr.	6 Hr.	15 Hr.	1 Day	2 Days	3 Days	
			14 hr.											
3	N.P.N....	54.5	111.0	..	..	..	..	157.7	..	..	166.6	..	69.2	Middle
	Chlorides	0.430	0.304	..	..	..	..	0.409	..	..	0.403	..	0.475	duodenum
			15 hr.											
7	N.P.N....	53.5	115.2	..	..	90.9	..	..	..	..	31.0	..	..	Middle
	Chlorides	0.415	0.391	..	..	0.426	..	..	..	..	0.315			duodenum
			26 hr.											
19	N.P.N....	38.0	103.4	128.5	..	..	..	..	..	..	94.2	..	..	Proximal
	Chlorides	0.389	0.346	0.485	..	..	..	..	..	..	0.452			jejunum
			24 hr.											
30	N.P.N....	39.0	103.8	..	86.3	..	..	..	..	..	..	..	..	Proximal
	Chlorides	0.481	0.409	..	0.448									jejunum
			16 hr.											
5	N.P.N....	66.6	105.0	84.5	..	..	..	..	..	..	30.0	..	..	18 inches
	Chlorides	0.468	0.462	0.485	..	..	..	..	..	..	0.498			jejunum
			22 hr.											
4	N.P.N....	44.5	267.8	..	..	..	215.9	..	..	176.5	..	..	44.9	18 inches
	Chlorides	0.436	0.330	..	..	..	0.397	..	..	0.462	..	..	0.495	jejunum
			43 hr.											
8	N.P.N....	39.5	214.3	..	..	..	..	..	225.4	..	..	285.7	..	Upper part
	Chlorides	0.482	0.339	..	..	..	..	..	0.403	..	..	0.426		of ileum
			43 hr.											
9	N.P.N....	42.2	178.6	..	..	..	..	..	..	..	..	138.2	..	Lower part
	Chlorides	0.455	0.419	..	..	..	..	..	..	..	..	0.508		of ileum

perature has at least approached normal; it bleeds freely and hops around in apparent health. The blood chlorides are well on the way to normal by the end of an hour after release of the obstruction, and within three or four hours they are at or above the normal level. The nonprotein nitrogen of the blood drops soon after release if the rabbit has not been moribund, but when it has been near death the nonprotein nitrogen may increase for a time after the release before there is a return toward normal. The return of the nonprotein nitrogen to normal after release of low obstructions is not so rapid as that after the release of high obstructions.

The removal of the stomach contents before the release of the obstruction retards recovery. Substitution of an equivalent amount of distilled water, although presumably compensating for the loss of fluid,

does not hasten recovery. The blood chlorides remain low, and the nonprotein nitrogen remains elevated until food or water containing chlorides is given. Substitution of an equivalent amount of sodium chloride solution to compensate for the water and chloride removed from the stomach before release of the obstruction hastens recovery more than when distilled water is given but does not permit of rapid return

TABLE 3.—*The Effect on Recovery of Removing Stomach Contents Before Release of Obstruction at the Third Inch of the Jejunum. The Changes Are Noted When Solutions of Sodium Chloride or Hydrochloric Acid Are Substituted for the Stomach Contents Removed Before Release of Obstruction*

Rabbit	Wt., Lb.		Before	Release 24 Hr.	2 Hr.	3 Hr.	18 Hr.	20 Hr.	42 Hr.	44 Hr.	
30	7½	N.P.N....	39.0	103.8	86.3	..	..	..	..	..	Stomach contents not removed; obstruction released; neither food nor water until after last blood was taken
		Chlorides	0.481	0.409	0.448						
27	...	N.P.N....	55.5	103.5	..	120.0	..	..	..	..	Stomach contents 153 Gm., 0.450 Gm. as sodium chloride. Stomach contents removed; obstruction released; neither food nor water until after last blood was taken
		Chlorides	0.505	0.420	..	0.413					
33	7¼	N.P.N....	62.3	160.0	192.0	..	..	201.0	..	247.0	Stomach contents 155 Gm., 0.580 Gm. as sodium chloride. Stomach contents removed; obstruction released; distilled water given freely after release until 20 hr., then food
		Chlorides	0.442	0.307	0.297	..	..	0.343	..	0.363	
42	5	N.P.N....	45.8	168.0	105.0	..	100.0	..	57.5	..	Stomach contents 160 Gm., 0.520 Gm. as sodium chloride. Stomach contents removed; obstruction released; 150 cc. water plus 0.6 Gm. sodium chloride given; distilled water 2 hr. after release, food 18 hr. after
		Chlorides	0.500	0.307	0.424	..	0.424	..	0.508		
41	7	N.P.N....	46.0	116.2	107.0	..	100.0	..	..	..	Stomach contents 215 Gm., 0.704 Gm. as sodium chloride. Stomach contents removed; obstruction released; 150 cc. water plus hydrochloric acid equivalent to 0.6 Gm. sodium chloride given; distilled water 2 hr. after release, food 18 hr. after
		Chlorides	0.500	0.370	0.439	..	0.508				

of blood chlorides and nonprotein nitrogen to normal. Substitution of a solution of hydrochloric acid equivalent to the water and chlorides removed before release induces recovery almost as rapidly as that occurring after release of obstruction without removal of the stomach contents.

Rabbits having had their stomach contents removed before release of obstruction without replacement of chlorides by sodium chloride or hydrochloric acid do not appear entirely healthy. They bleed with

difficulty and are sluggish in their movements until they have been given food or water and their blood constituents return toward a normal level.

Comment.—The experimental observations reveal certain facts concerning intestinal obstruction, a few of which are new. They also justify explanations that may or may not be substantiated.

Rabbits die from obstruction of much shorter duration than do dogs. One rather obvious reason is that there is no vomiting and therefore no release of pressure in the obstructed bowel. Although rabbits do not vomit, the retained secretions are lost to the animal just as much as if vomiting had occurred. The amount of chloride in the contents of the obstructed bowel is sufficient to account for the fall in blood chlorides.

The higher the obstruction, up to the middle of the duodenum, the more rapid the death. It has been proved for rabbits and apparently holds true for dogs that the stomach and duodenum receive practically all the secretions entering the gastro-intestinal tract. These secretions come from the glands in the mucosa of the stomach and duodenum and also from the liver and pancreas.[5] Loss of all the secretions entering the stomach and duodenum is incompatible with life. The lower the obstruction, the more reabsorption of these secretions, thus retarding dehydration as well as depleting the elements in blood going to form the secretions. During the first twenty-four hours of ileal obstruction there is almost no evidence of change in a rabbit's condition. The blood chloride, nonprotein nitrogen and alkali reserve differ but little from the normal. The animal appears healthy; it is warm and bleeds from its veins readily. During this first twenty-four hour interval reabsorption almost keeps pace with secretion. Since the entire intestinal tract is not available for absorption and none of the secreting portion is excluded, absorption lags behind secretion. Furthermore, as intraintestinal pressure is slowly increased, peristalsis is more vigorous, and secretion is accelerated. The increased amount of secretion augments intraintestinal pressure, giving rise to distention as the intestinal muscles lose their tone. As distention increases, absorption is proportionately retarded and is eventually almost nil. The changes in the blood during this period are great, and the change in the clinical picture is evident. All determinable deviations from normal are the same as those occurring in high obstruction but occur from twenty-four to forty-eight hours later.

In spite of apparent evidence to the contrary, it may be that toxemia plays a minor rôle in simple obstruction and that the real cause of death is the loss of secretions normally reabsorbed. The fact that extirpation of the duodenum has been found compatible with life need

5. Bunting and Jones: J. Exper. Med. **17**:192, 1913; **18**:25, 1913.

not alter this conception, for then there is no loss of secretions, the elements forming the secretions never being removed from the blood.

The release of temporary obstructions in rabbits, even when the animal is near death, permits return to health. The reabsorption of the contents of the obstructed bowel through normal intestine below the obstruction or from the obstructed bowel after the tension is lowered instead of showing evidence of the absorption of a noxious substance initiates a rapid return of the animal to normal. The blood chlorides are increased within one hour after release of the obstruction. This increase must be due to reabsorption, for the removal of the bowel contents above the obstruction prior to release does not permit an increase in the blood chlorides. This observation is further confirmation of the concept that the decrease in blood chlorides in obstruction is due solely to loss of stomach secretions. Furthermore, unless chlorides are given, the high nonprotein nitrogen shows no inclination to decrease. Some animals have lived for almost two days after release of jejunal obstructions with low blood chlorides and high nonprotein nitrogen and eventually recovered after the ingestion of food and water containing chlorides.

The removal of the stomach contents prior to release of temporary obstruction and the substitution of solutions of sodium chloride or hydrochloric acid in amounts comparable to the amount of water and chloride removed permit of recovery more rapidly than when distilled water is substituted but not so rapidly as when the obstruction is released without removal of the stomach contents. There are other elements in the secretions besides chlorides, the reabsorption of which benefits the animal.

THE EFFECTS OF DIMINISHED AND INCREASED INTRA-INTESTINAL PRESSURE

Attempts to reduce the intra-intestinal pressure in obstructed bowel were not uniformly successful and entailed additional operative trauma, which must be considered in interpreting results.

Jejunostomy, theoretically, causes only the entire loss of the secretions from the liver, pancreas and the gastro-intestinal mucosa above the segment opened. In the jejunostomies performed an element of partial obstruction was added in spite of efforts to prevent it, for none of the rabbits drained freely enough to obviate some duodenal distention. This presumably shortened the lives of the animals but does not entirely vitiate the observation that the length of life following jejunostomy is but little longer than that following obstruction at the same point. The changes in the blood are identical, and the appearance of the animal is the same.

Removal of the stomach contents every eight hours from an animal whose jejunum is obstructed relieves most of the excess intra-intestinal

pressure, although the duodenum is always moderately distended. This incomplete release of pressure in jejunal obstructions increases the time of survival from eight to fifteen hours.

Reducing intraduodenal pressure in jejunal obstruction by gastrostomy, done at the time the obstruction was created, adds a great deal of operative trauma but, even so, lengthens life from ten to fifteen hours.

The capacity of the stomach, or more likely the duodenum, to absorb water is much greater than has been supposed. Attempts to increase extra-intestinal pressure after obstruction of the jejunum by the introduction of water into the stomach in fairly large amounts were not

TABLE 4.—*The Effects of Reducing Intra-intestinal Pressure on Length of Life*

Rabbit		Before	20 Hr.	24 Hr.	25½ Hr.	26 Hr.	30 Hr.	44 Hr.	46 Hr.	
10	N.P.N....	44.4	78.9	..	..	..	..	..	..	Obstruction third inch of jejunum; not very sick at 20 hr.
	Chlorides	0.495	0.460							
25	N.P.N....	48.7	..	..	..	111.5	..	..	..	Obstruction third inch of jejunum; fairly sick at 26 hours.
	Chlorides	0.487	..	..	..	0.349				
16	N.P.N....	43.1	..	..	188.0	..	..	..	..	Jejunostomy third inch of jejunum; fairly sick; killed
	Chlorides	0.420	..	..	0.317	..	..	..	..	
74	N.P.N....	36.2	..	109.2	..	..	154.0	..	..	Obstruction third inch of jejunum; stomach contents removed 8, 16 and 24 hours after obstruction; fairly sick; killed at 30 hours
	Chlorides	0.439	..	0.401	..	..	0.396	..	..	
94	N.P.N....	40.5	..	58.0	..	..	..	..	48.7	Stomach sutured to anterior belly wall as control; lived
	Chlorides	0.462	..	0.462	..	..	..	..	0.479	
96	N.P.N....	50.0	..	103.6	..	..	..	243.9	..	Pyloric obstruction and gastrostomy permitting complete emptying of stomach; dying at 44 hours
	Chlorides	0.420	..	0.366	..	..	..	0.257	..	
92	N.P.N....	46.1	..	152.6	..	..	..	..	..	Obstruction third inch of jejunum and gastrostomy permitting complete emptying of stomach; died at 33 hours
	Chlorides	0.515	..	0.469	..	..	..	..	..	

successful. The excess water was readily absorbed, for when the animal was killed, there were but little more stomach and duodenal contents than are found in rabbits of the same size after simple obstruction. This work was done during hot weather which probably influenced the results.

The introduction of liquid albolene into the stomach after obstruction of the jejunum gave different results. Since there was no absorption of albolene, intra-intestinal pressure was increased immediately, and death occurred from eight to ten hours sooner than it does after simple obstruction. There was also an increased amount of stomach and duodenal contents, rabbits so treated having about the same amount (albolene discarded) at their death in twenty hours as those with simple obstruction had in from twenty-six to thirty hours. The amount of chlorides in the stomach contents was virtually the same in the two groups.

Increasing intra-intestinal pressure after jejunal obstruction by the introduction into the stomach of stomach and duodenal contents, obtained after twenty-six hours of jejunal obstruction in another animal, shortened the usual length of life and caused a greater increase in the nonprotein nitrogen of the blood than that observed after using albolene.

Since the duration of life is longer after ileal obstruction, increments in intra-intestinal pressure make differences in the time of survival more

TABLE 5.—*The Effects of Increasing Intra-intestinal Pressure in Animals Obstructed at the Third Inch of the Jejunum. The Pressure Was Increased by Giving Water, Liquid Albolene or the Stomach Contents of a Rabbit Having Had a Jejunal Obstruction for Twenty-Six Hours Through a Catheter Passed into the Stomach*

Rabbit		Before	19 Hr.	20 Hr.	22 Hr.	24 Hr.	30 Hr.	
10	Nonprotein nitrogen	44.4	..	78.9	..	..	..	Obstructed third inch of jejunum; not very sick
	Chlorides	0.495	..	0.460				
27	Nonprotein nitrogen	55.5	..	..	..	103.5	..	Obstructed third inch of jejunum; fairly sick
	Chlorides	0.505	..	..	..	0.420	..	
64	Nonprotein nitrogen	48.0	..	..	..	77.7	..	Obstructed third inch of jejunum; 100 cc. water immediately; not very sick; killed in 24 hours
	Chlorides	0.515	..	..	..	0.363	..	
76	Nonprotein nitrogen	39.9	..	..	..	129.8	157.3	Obstructed third inch of jejunum; 100 cc. water immediately, 100 cc. in 8 hours, 75 cc. in 24 hours; very sick; killed in 30 hours
	Chlorides	0.427	..	..	..	0.391	0.549	
85	Nonprotein nitrogen	53.7	..	..	122.0	..	..	Obstructed third inch of jejunum; 120 cc. albolene immediately; fairly sick; killed in 22 hours
	Chlorides	0.467	..	..	0.430	..	..	
77	Nonprotein nitrogen	58.8	200.0	..	..	..	..	Obstructed third inch of jejunum; 100 cc. albolene immediately, 100 cc. in 7 hours; dying, killed in 19 hours
	Chlorides	0.502	0.346					
86	Nonprotein nitrogen	49.6	..	..	177.0	..	..	Obstructed third inch of jejunum; 120 cc. stomach contents immediately; fairly sick; killed in 22 hours
	Chlorides	0.512	..	..	0.429	..	..	
87	Nonprotein nitrogen	60.0	..	..	203.0	..	..	Obstructed third inch of jejunum; 120 cc. stomach contents immediately; fairly sick; killed in 22 hours
	Chlorides	0.508	..	..	0.430	..	..	

apparent. The capacity of the small intestine for the absorption of water is great. Rather large amounts of water introduced into the stomach at short intervals following obstruction at the terminal ileum caused no appreciable increase in intra-intestinal pressure and did the animals no harm. The work was done during hot weather which probably affected the results.

Increasing intra-intestinal pressure by the introduction of liquid albolene into the stomach after obstruction at the terminal ileum shortens life materially. The animals die with as great changes in their blood chlorides and nonprotein nitrogen after the same duration of obstruction as those having high jejunal obstructions.

The effects of the introduction of stomach and duodenal contents, obtained from an animal having jejunal obstruction for twenty-six hours, into the stomach of a rabbit immediately following obstruction at the terminal ileum are not great. The animals are but little more distended at the end of twenty-four hours than are those having simple ileal obstruction, and the deviations of their blood chlorides and nonprotein nitrogen from the normal are not much greater.

Comment.—Reducing the pressure that develops within the bowel above the level of the induced obstruction increases the duration of survival and restricts correspondingly the alterations in blood chlorides

TABLE 6.—*The Effects of Increasing Intra-intestinal Pressure in Rabbits Obstructed at the Terminal Ileum. The Pressure was Increased by Giving Water, Liquid Albolene or the Stomach Contents of a Rabbit Having Had a Jejunal Obstruction for Twenty-Six Hours, Through a Catheter Passed into the Stomach*

Rabbit		Before	22½ Hr.	24 Hr.	43 Hr.	
9	Nonprotein nitrogen	42.2	..	..	173.3	Ileal obstruction; fairly sick
	Chlorides	0.455	..	..	0.419	
63	Nonprotein nitrogen	39.2	..	52.2	..	Ileal obstruction; 100 cc. water immediately; not sick
	Chlorides	0.521	..	0.452		
69	Nonprotein nitrogen	46.8	..	70.0	..	Ileal obstruction; 100 cc. water immediately and at 2, 4 and 6 hours; not much distention; not sick
	Chlorides	0.500	..	0.372		
89	Nonprotein nitrogen	48.3	166.6	..	..	Ileal obstruction; 180 cc. albolene immediately, 100 cc. in 8 hours; enormous distention; dying in 22½ hours
	Chlorides	0.508	0.369			
91	Nonprotein nitrogen	50.1	157.7	..	..	Ileal obstruction; 180 cc. albolene immediately, 100 cc. in 8 hours; very sick; greatly distended; killed in 22½ hours
	Chlorides	0.512	0.438			
90	Nonprotein nitrogen	46.1	87.3	..	..	Ileal obstruction; 180 cc. stomach contents immediately, 100 cc. in 6 hours; moderately sick; moderately distended; killed in 22½ hours
	Chlorides	0.495	0.475			
103	Nonprotein nitrogen	46.0	116.0	..	..	Ileal obstruction; 180 cc. stomach contents immediately, 100 cc. in 5 hours; not very sick; not much distention; killed in 22½ hours
	Chlorides	0.475	0.424			

and nonprotein nitrogen. The reasons are definite. As intra-intestinal pressure rises above normal, hyperperistalsis is provoked and continues until the increasing fatigue of the muscularis results in corresponding grades of distention. The rate of secretion is in general directly proportional to the activity of peristalsis. The rate of absorption is inversely proportional to the degree of distention. Thus it follows that increasing intra-intestinal pressure provokes hyperperistalsis which in turn stimulates secretion; the resulting distention diminishes the capacity for reabsorption of these secretions.

The following observation is illustrative. Obstruction of the jejunum was made in the same manner in rabbits of the same size and continued for twenty-six hours. If the stomach contents are removed by aspiration

every eight hours, the total amount obtained is less than when the contents are allowed to collect under increasing pressure for the full period of the obstruction. Longer life after obstruction and restricted alterations in blood chlorides and nonprotein nitrogen provided by reducing intra-intestinal pressure cannot be the result of a decreased absorption of a toxic substance from the lumen of the bowel. The rate of absorption is augmented by reducing tension, and, if an absorbable toxic substance is present, life would be shortened and the symptoms of intoxication would be intensified. Available evidence indicates that survival is longer when intra-intestinal pressure is reduced because there is not so great a loss of the constituents forming the secretions that enter the stomach and duodenum.

The increase of intra-intestinal pressure in jejunal obstruction induced by the introduction of 120 cc. of albolene into the stomach stimulates secretion and retards reabsorption. Blood chlorides are just as low at the end of nineteen hours as they are after twenty-six hours when the pressure is not thus increased. The distention in itself is not the cause of early death, for these animals are greatly distended immediately after obstruction. They seem perfectly healthy until a few hours before death when the typical lethal symptoms develop.

The greater deviation from the normal level of the nonprotein nitrogen of the blood noted after increasing intra-intestinal pressure by introducing 120 cc. of the contents of obstructed bowel from another animal is difficult to explain. Clinically, these animals appeared no worse than those given albolene. It is possible that a larger series would show different results. The only plausible explanations are the presence of a noxious product in the stomach and duodenal contents introduced or that the contents introduced are sufficiently irritating to stimulate secretion more than does albolene.

The untoward effect of increasing intra-intestinal pressure following obstruction at the terminal ileum is evident. When pressure is not increased, there is an interval of about twenty-four hours in the animals with low obstructions before reabsorption lags too far behind secretion. The introduction of liquid albolene immediately after the obstruction is produced seems almost entirely to inhibit reabsorption as well as indirectly to stimulate secretion so that the animals die almost as quickly as do those having duodenal obstructions. The fact that the blood chlorides and nonprotein nitrogen are approximately the same as those found after the same duration of duodenal obstruction makes it evident that death is not due solely to distention. Furthermore, even though greatly distended immediately after obstruction, they appear to be healthy until a few hours before death.

The effects of increasing intra-intestinal pressure following obstructions at the terminal ileum by introducing contents of obstructed bowel into the stomach are not great. Death occurs little sooner than it

does following simple ileal obstruction. At the end of twenty-four hours, the changes of the blood chloride and of the nonprotein nitrogen from the normal are not marked. There is little distention, since the water in the obstructed contents is absorbed.

The difference in the effect produced by introducing the stomach and duodenal contents into the stomach to increase pressure in jejunal and ileal obstructions may be explained by the rapid descent of the contents in ileal obstruction to the jejunum and ileum where they cannot stimulate secretion by irritation of the stomach and duodenal mucosa.

TABLE 7.—*The Therapeutic Value of Solutions of Sodium Chloride as Compared to Dextrose Given Intravenously When Intra-intestinal Pressure Is Not Diminished. Stomach and Duodenal Contents Removed After Death and Analyzed for Chloride Content*

Rabbit	Wt., Lb.		Before	20 Hr.	24 Hr.	26 Hr.	
71	4½	Nonprotein nitrogen	41.3	..	152.6	..	Obstructed third inch jejunum
		Chlorides	0.488	..	0.420		
		Alkali reserve	..	..	69.0		
78	4½	Nonprotein nitrogen	38.9	..	142.0	..	Obstructed third inch jejunum; contents of obstructed bowel, 222 Gm., 0.741 Gm. as sodium chloride
		Chlorides	0.488	..	0.396		
58	5½	Nonprotein nitrogen	42.5	152.6	..	..	Obstructed third inch jejunum; 40 cc. 5 per cent dextrose at 8 and 16 hours after obstruction
		Chlorides	0.528	0.380			
		Alkali reserve	..	65.5			
59	6	Nonprotein nitrogen	48.7	132.4	..	..	Obstruction third inch jejunum; 40 cc. 0.9 per cent sodium chloride at 8 and 16 hours after obstruction
		Chlorides	0.503	0.538			
		Alkali reserve	..	58.0			
46	4½	Nonprotein nitrogen	38.0	..	..	125.3	Obstructed third inch jejunum; 40 cc. 5 per cent dextrose at 8 and 16 hours after obstruction; contents of obstructed bowel 278 Gm., 0.884 Gm. as sodium chloride
		Chlorides	0.475	..	..	0.353	
47	3¾	Nonprotein nitrogen	53.0	..	..	124.5	Obstructed third inch jejunum; 40 cc. 0.9 per cent sodium chloride at 8 and 16 hours after obstruction; contents of obstructed bowel 251 Gm., 1.082 Gm. as sodium chloride
		Chlorides	0.473	..	..	0.538	

THE THERAPEUTIC VALUE OF SOLUTIONS OF SODIUM CHLORIDE

The intravenous administration of solutions of 5 per cent dextrose or of 0.9 per cent sodium chloride every eight hours after jejunal obstruction lengthens the life of rabbits from two to six hours. There seems to be no difference between the effects of the two solutions as far as survival is concerned. There is with both an increase in the amount of distention due primarily to increased secretion and finally to diminished absorption as distention progresses. The partial correction of the developing dehydration enables the glands of the mucosa to secrete more than when no fluids are given. The harmful effects of the increased intra-intestinal pressure are a little more than offset by the beneficial effects of diminished dehydration. Solutions of sodium chloride seem to have no specific curative effect. The loss of chlorides into the contents

of the obstructed bowel is compensated for by that given intravenously, but the nonprotein nitrogen increases almost as much as when solutions of dextrose are given.

When intra-intestinal pressure is reduced by aspiration or gastrostomy or when all secretions are lost with a jejunostomy, the therapeutic value of solutions of sodium chloride given intravenously or

TABLE 8.—*The Therapeutic Value of Solutions of Sodium Chloride as Compared to Dextrose Given Intravenously When Intra-intestinal Pressure Is Reduced by the Methods Indicated. The Stomach Contents Were Removed Through a Catheter Passed into the Stomach*

Rabbit	Wt., Lb.	Before	24 Hr.	28 Hr.	30 Hr.	36 Hr.	37 Hr.	38 Hr.	47 Hr.	48 Hr.	67 Hr.	
74	4½	..	..	..	..	..	..	..	..	..	..	Obstructed third inch jejunum; stomach contents removed 8, 16, 24 and 30 hours after obstruction
N.P.N....		36.2	109.2	..	154.0							
Chlorides		0.439	0.401	..	0.396							
36	4¾	..	..	..	..	..	..	Died	..	..	..	Obstructed third inch jejunum; 40 cc. 0.9 per cent sodium chloride 8, 16, 24 and 32 hours after obstruction; stomach contents removed 8, 16, 24 and 32 hours after obstruction
N.P.N....		40.0	104.0									
Chlorides		0.491	0.479									
43	...	..	..	..	..	Died	..	..	..	..	..	Jejunostomy; 40 cc. 0.9 per cent sodium chloride 8 and 16 hours after operation; partial obstruction
N.P.N....		53.0	96.6									
Chlorides		0.496	0.545									
44	...	..	..	..	..	..	Dying	..	..	..	..	Jejunostomy; 40 cc. 5 per cent dextrose 8 and 16 hours after operation
N.P.N....		58.8	148.8	..	..	..	274.0					
Chlorides		0.512	0.429	..	..	..	0.375					
82	...	..	..	..	..	..	..	..	Dying	..	..	Jejunostomy, 2 stage; 40 cc. 0.9 per cent sodium chloride 8, 16, 24, 32 and 40 hours after operation
N.P.N....		58.8	112.3	..	..	..	..	..	224.3			
Chlroides		0.508	0.578	..	..	..	..	..	0.548	..	..	
93	...	..	..	..	..	Dying	..	..	..	..	..	Obstructed third inch jejunum; gastrostomy; 40 cc. 2.5 per cent dextrose 8, 16, 24 and 32 hours after obstruction
N.P.N....		44.0	157.4	..	..	208.3						
Chlorides		0.459	0.452	..	..	0.376						
98	...	..	..	Dying	..	..	..	..	..	..	..	Pyloric obstruction; gastrostomy; 40 cc. 2.5 per cent dextrose 7, 13 and 23 hours after obstruction
N.P.N....		46.8	..	240.9								
Chlorides		0.462	..	0.370								
101	...	..	..	..	..	..	..	..	..	..	Dying	Pyloric obstruction; gastrostomy; 40 cc. 0.9 per cent sodium chloride 7, 13, 23, 30, 35, 46, 52 and 58 hours after obstruction
N.P.N....		45.4	..	..	..	..	..	..	..	145.9	229.8	
Chlorides		0.488	..	..	..	..	..	..	..	0.393	0.343	

subcutaneously becomes evident. Life is prolonged from twelve to sixteen hours by the administration of sodium chloride solution at short intervals if the stomach contents are removed every eight hours from an animal with jejunal obstruction. There is less increase in the nonprotein nitrogen in the blood than when solutions of dextrose are given.

The difference in value between the two solutions is evident when they are given to animals on which a jejunostomy has been performed.

The duration of life is shorter, and the nonprotein nitrogen is higher when dextrose is administered than when saline is used.

Reduction of intra-intestinal pressure in jejunal obstruction by means of gastrostomy adds considerable operative trauma so that the results are undeterminate. Presumably life would have been prolonged considerably beyond the actual time noted if this trauma had not been added.

The difference of twenty hours in the length of survival of animals suffering loss of both stomach and duodenal secretions as found in those having a jejunostomy, and a loss of only stomach secretions as seen in pyloric obstruction with gastrostomy is evidence that the loss of duodenal secretions is not compensated for by saline. This differ-

TABLE 9.—*The Effect of Intravenous Administration of the Solutions Indicated on the Alkali Reserve and Nonprotein Nitrogen of Blood*

Rabbit	Wt., Lb.		Before	24 Hr.	
56	5	Normal animal.....	..	..	Starved 24 hours; water given freely
		Alkali reserve.....	..	56.4	
52	5½	Nonprotein nitrogen	37.0	41.0	Starved 72 hours; water given freely; given 30 cc. 2.5 per cent dextrose in vein at beginning of experiment and again 10 hours later; rabbit bled from heart and killed in 24 hours; distilled water given freely
		Chlorides.....	0.492	0.517	
		Alkali reserve.....	..	61.4	
53	4¾	Nonprotein nitrogen	32.0	42.0	Starved 72 hours; water given freely; given 20 cc. 3 per cent sodium bicarbonate neutralized with lactic acid in vein at beginning of experiment and again 10 hours later; rabbit bled from heart and killed in 24 hours; distilled water given freely
		Chlorides.....	0.479	0.462	
		Alkali reserve.....	..	65.4	
54	5½	Nonprotein nitrogen	35.7	46.5	Starved 72 hours; water given freely; given 20 cc. 1 per cent lactic acid in vein at beginning of experiment and again 10 hours later; rabbit bled from heart and killed in 24 hours; distilled water given freely
		Chlorides.....	0.492	0.465	
		Alkali reserve.....	..	54.0	
50	2½	Nonprotein nitrogen	33.5	62.8	No starvation; given 20 cc. 3 per cent sodium bicarbonate in vein at beginning of experiment and again 10 hours later; rabbit bled from heart and killed in 24 hours; distilled water given freely
		Chlorides.....	0.462	0.412	
		Alkali reserve.....	..	95.0	

ence in survival would probably have been still greater had sufficient sodium chloride been given the animal with pyloric obstruction to maintain the blood chloride at the normal level.

The intravenous administration of solutions of dextrose or of sodium bicarbonate neutralized with lactic acid caused no appreciable changes in the alkali reserve or nonprotein nitrogen of a healthy rabbit.

Sufficient lactic acid to produce noticeable changes in the alkali reserve could not be given without immediately killing the animal.

Solutions of sodium bicarbonate given intravenously in sufficient quantity materially to increase the alkali reserve caused a great increase in the nonprotein nitrogen of the blood.

Comment.—The therapeutic value of the intravenous or subcutaneous administration of solutions of sodium chloride increases as

intra-intestinal pressure is diminished and, conversely, the value decreases as intra-intestinal pressure is increased. The increased supply of fluid enables the animal to increase its gastric and duodenal secretions, thus augmenting distention and diminishing reabsorption unless means are employed to reduce intra-intestinal pressure. The value of sodium chloride lies in the establishment of a normal blood chloride level which corrects alkalosis and thereby lowers the high nonprotein nitrogen.

Life is prolonged most by sodium chloride in pyloric obstructions when gastric distention is prevented by gastrostomy. Here there is loss of gastric secretions of which the chloride is the most important constituent. Replacement of this chloride loss would presumably permit of survival until starvation occurred were it not for the operative trauma. Continued removal of accumulated secretions in the stomach from rabbits with jejunal obstruction and the administration of sodium chloride solutions almost doubles the length of life of rabbits as compared with those having simple jejunal obstruction.

The kidneys show little damage from intestinal obstruction. There is an increased output of urea in the urine during intestinal obstruction as well as the increase in the blood urea.[6] Evidently catabolic processes are forming waste products more rapidly than normal and more rapidly than the kidney can excrete the accumulating urea. The diminished chlorides may be the cause of this increased catabolism. Presumably there is no appreciable damage to the kidney following twenty-four hour alkalosis induced by the intravenous administration of sodium bicarbonate. The increased nonprotein nitrogen in the blood must then be accounted for by increased catabolism or by abnormal metabolism. The alkalosis accompanying intestinal obstruction may be the cause of part of the increase found in the nonprotein nitrogen. Such an explanation accounts for the rapid decrease in the nonprotein nitrogen found when solutions of sodium chloride are given to correct the alkalosis.

CLINICAL APPLICATION

Rabbits differ from human beings in many respects; nevertheless, certain conclusions seem warranted.

The administration of isotonic or slightly hypertonic solutions of sodium chloride is of great value when measures are taken to insure the least possible increase above the normal intra-intestinal pressure. These measures include the release of the obstruction. The intestinal musculature in human beings does not regain its tone after distention so rapidly as in rabbits, so that simply releasing the obstruction may not reduce intra-intestinal pressure. When the obstruction is high, gastric lavage may diminish pressure sufficiently to restore muscle tone.

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6. Haden, R. L., and Orr, T. G.: Bull. Johns Hopkins Hosp. **34**:26, 1923.

When a large part of the intestine is distended, it must be emptied, preferably by a method causing the least trauma. The intestinal contents need not be removed because of their toxicity, but they must be expelled to relieve pressure. In some instances the intravenous administration of 25 per cent sodium chloride will stimulate peristalsis sufficiently to induce propulsion of intestinal contents and relieve undue pressure. It may be necessary to remove the contents mechanically as by aspiration with trocars at numerous levels. This procedure, in spite of all precautions, is likely to soil the peritoneal cavity and is accompanied by the deleterious effects of trauma. A jejunostomy may be done, not to drain away all the secretions, but to relieve pressure sufficiently to permit of reabsorption of secretions. When intraintestinal pressure is low or has been reduced, it can be kept low by measures that diminish secretion. Large doses of morphine inhibit peristalsis and diminish secretion and thereby aid materially in keeping intra-intestinal pressure low. Such measures will insure the greatest amount of reabsorption of secretions, and thus will aid materially in promoting recovery.

Peritonitis is, in most instances, more dangerous because of the accompanying intestinal paresis than because of the toxicity of infection. Similar methods of treatment are applicable. When there is little distention, the withholding of nourishment by the mouth diminishes secretion. Morphine in liberal doses further decreases secretion so that the limited capacity for absorption is not overtaxed. Solutions of sodium chloride and of dextrose given intravenously restore any deficiency of blood chlorides to normal as well as supply nourishment.

When there is considerable distention, jejunostomy will aid in reducing intra-intestinal pressure. The jejunostomy should be done so as to relieve excess pressure but not to afford complete drainage of gastric and duodenal secretions, for such complete drainage is as disastrous as continued paresis.

SUMMARY

1. Rabbits recover more rapidly from intestinal obstruction if they are permitted to reabsorb the contents of obstructed intestine than when such contents are removed.

2. Diminishing intra-intestinal pressure in obstructed bowel prolongs the life of rabbits, because it diminishes secretions and promotes reabsorption.

3. Increasing the intra-intestinal pressure in obstructed bowel shortens the life of rabbits, because it stimulates secretion and diminishes reabsorption.

4. The therapeutic value of solutions of sodium chloride increases as intra-intestinal pressure in the obstructed bowel diminishes, and, conversely, the value diminishes as intra-intestinal pressure increases.

PROTOCOLS: PART I

Rabbit 1—Duodenal obstruction—1/24/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours after Obstruction	48 Hours after Obstruction
Nonprotein nitrogen	53.5	50.5	50
Chlorides	0.353	0.370	0.383

Ethylene anesthesia 10 minutes. Obstruction, rubber band, middle duodenum. Blood from ear. Water ad lib postoperatively. Killed after 48 hours. Obstruction not complete.

Rabbit 2—Duodenal obstruction—1/24/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	12 Hours after Obstruction	5 Hours after Release
Nonprotein nitrogen	82.1	224.5	242
Chlorides	0.413	0.355	0.424

Ethylene anesthesia 12 minutes. Obstruction, rubber band, middle duodenum. Blood from ear. Bled with difficulty after 12 hours. Rabbit cold. Obstruction released in 12 hours. Water ad lib postoperatively. Food 5 hours after release at which time rabbit seemed perfectly healthy. Living and apparently healthy 1 week later.

Rabbit 3—Duodenal obstruction—1/31/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	14 Hours After Obstruction	4½ Hours After Release	29 Hours After Release	3 Days After Release
Nonprotein nitrogen	54.5	111	157.7	166.6	69.2
Chlorides	0.430	0.304	0.469	0.403	0.475

Ethylene anesthesia 12 minutes. Obstruction, rubber band, middle duodenum. Blood from ear. Obstruction released after 14 hours at which time rabbit was bled with difficulty. Water ad lib postoperatively. Food 4½ hours after release at which time rabbit appeared perfectly healthy. Living and apparently healthy 1 week later.

Rabbit 4—Jejunal obstruction—1/31/27. General diet: No food for 48 hours; water ad lib

	Before Obstruction	22 Hours After Obstruction	29 Hours After Release	3½ Hours After Release	15 Hours After Release	3 Days After Release
Nonprotein nitrogen	44.5	178.5	267.8	245.9	176.5	44.9
Chlorides	0.436	0.399	0.360	0.397	0.462	0.495

Ethylene anesthesia 10 minutes. Obstruction, rubber band, 18 inches below beginning of jejunum. Blood from ear. Obstruction released after 29 hours. Water ad lib postoperatively. Food 3½ hours after release. Living and apparently healthy 1 week later.

Rabbit 5—Jejunal obstruction—2/7/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	16 Hours After Obstruction	1 Hour After Release	24 Hours After Release
Nonprotein nitrogen	66.6	105	84.5	30
Chlorides	0.468	0.462	0.485	0.498

Ethylene anesthesia 10 minutes. Obstruction, rubber band, 18 inches below beginning of jejunum. Blood from ear. Obstruction released in 16 hours. Water ad lib postoperatively. Food 1 hour after release. Living and apparently healthy 1 week later.

Rabbit 6—Jejunal obstruction—2/7/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	16 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen	42.9	137.9	115.4
Chlorides	0.482	0.405	0.482

Ethylene anesthesia 10 minutes. Obstruction, rubber band, 18 inches below beginning of jejunum. Blood from ear. Obstruction released in 16 hours. Water ad lib postoperatively. Food 2 hours after release. Living and apparently healthy 1 week later.

Rabbit 7—Jejunal obstruction—2/7/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	15 Hours After Obstruction	3 Hours After Release	24 Hours After Release
Nonprotein nitrogen	53.5	115.2	90.9	31
Chlorides	0.415	0.391	0.420	0.515

Ethylene anesthesia 10 minutes. Obstruction, rubber band, 18 inches below beginning of jejunum. Blood from ear. Obstruction released in 15 hours. Water ad lib postoperatively. Food 3 hours after release. Living and apparently healthy 1 week later.

Rabbit 8—Ileal obstruction—2/24/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	43 Hours After Obstruction	6 Hours After Release	2 Days After Release
Nonprotein nitrogen.....	39.5	214.3	225.4	285.7
Chlorides	0.482	0.339	0.403	0.426

Ethylene anesthesia 8 minutes. Obstruction, rubber band, upper ileum. Blood from ear. Obstruction released in 43 hours. Water ad lib postoperatively. Food 6 hours after release. Rabbit died 4 days after obstruction from pneumonia.

Rabbit 9—Ileal obstruction—2/24/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	43 Hours After Obstruction	49 Hours After Obstruction	2 Days After Release
Nonprotein nitrogen	42.2	173.3	178.6	138.2
Chlorides	0.455	0.419	.....	0.508

Ethylene anesthesia 8 minutes. Obstruction, rubber band, terminal ileum. Blood from ear. Obstruction released in 49 hours. Water ad lib postoperatively. Food immediately after release. Rabbit died 1 week after release from adhesions producing partial intestinal obstruction.

Rabbit 10—Jejunal obstruction—3/8/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	20 Hours After Obstruction	5 Hours After Release	40 Hours After Release
Nonprotein nitrogen.....	44.4	78.9	123.7	39.6
Chlorides	0.495	0.460	0.475	0.532

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Obstruction released in 20 hours. Water ad lib postoperatively. Food 5 hours after release. Living and apparently healthy 1 week later.

Rabbit 11—Jejunal obstruction—3/8/27. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	43.4	133.1
Chlorides	0.502	0.409

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Obstruction released in 24 hours. Water ad lib postoperatively. Food immediately after release. Living and apparently healthy 1 week later.

Rabbit 13—Control operation—3/29/27. General diet: No food for 48 hours; water ad lib.

	Before Operation	22 Hours After Operation	49 Hours After Operation
Nonprotein nitrogen	37	47.9	38.2
Chlorides	0.466	0.461	0.471

Ethylene anesthesia 10 minutes. Abdomen was opened, intestines drawn out of abdominal cavity and replaced. There was considerably more manipulation of the intestines than usually occurs in producing obstruction. Blood from ear. Rabbit made an uneventful recovery.

Rabbit 14—Jejunal obstruction—11/1/27. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	44.1	122.4
Chlorides	0.432	0.376

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	157.8	0.206	0.325

Ethylene anesthesia 10 minutes. Obstruction, umbilical tape, third inch jejunum. Blood from ear. Rabbit killed 24 hours after obstruction. Neither food nor water postoperatively. There were no signs of severe toxemia. Stomach and duodenal contents removed.

Rabbit 15—Jejunal obstruction—11/1/27. Weight 3½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	48.3	145.1
Chlorides	0.429	0.370

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	191.7	0.249	0.479

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, third inch jejunum. Blood from ear. Rabbit killed in 24 hours. Neither food nor water postoperatively. No signs of severe toxemia. Stomach and duodenal contents removed.

Rabbit 18—Jejunal obstruction—11/21/27. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen	32.4	120
Chlorides	0.393	0.346

Ethylene anesthesia 12 minutes. Obstruction, umbilical tape, third inch jejunum. Blood from ear. Rabbit killed in 26 hours after obstruction. Neither food nor water postoperatively.

Rabbit 19—Jejunal obstruction—11/21/27. Weight 4¼ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction	1 Hour After Release	24 Hours After Release
Nonprotein nitrogen	38	103.4	128.5	94.2
Chlorides	0.389	0.346	0.485	0.453

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Obstruction released in 26 hours. Neither food nor water postoperatively, until 24 hours after release. Living and apparently healthy 1 week later.

Rabbit 20—Control—12/12/27. Weight 5¼ pounds. General diet: No food for 48 hours; water ad lib.

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	153.5	0.310	0.476
Intestinal	117.5	0.060	0.071

Normal rabbit killed. Stomach and duodenal contents removed. Small and large intestinal contents removed.

Rabbit 21—Jejunal obstruction—12/20/27. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction	2 Hours After Release	24 Hours After Release
Nonprotein nitrogen	51.8	186.2	180.7	187
Chlorides	0.436	0.435	0.410	0.485

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Obstruction released in 26 hours. Neither food nor water postoperatively until 2 hours after release. Living and apparently healthy 1 week later.

Rabbit 22—Jejunal obstruction—12/20/27. Weight 5¼ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen	48.7	213.4
Chlorides	0.446	0.420

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	192	0.212	0.407
Intestinal	60	0.156	0.105

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. First blood from ear. Second from heart. Rabbit too sick to bleed at 26 hours. Killed and blood taken from heart. Stomach and duodenal contents removed. Small and large duodenal contents removed. Neither food nor water postoperatively. A great deal of gas in stomach and duodenum causing enormous distention.

Rabbit 23—Control—12/28/27. General diet.

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	139	0.457	0.635
Intestinal	61.5	0.045	0.028

Normal rabbit killed after regular feeding. Stomach and duodenal contents removed. Small and large intestinal contents removed.

Rabbit 24—Jejunal obstruction—2/9/28. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction	1½ Hours After Release
Nonprotein nitrogen	46.1	109.5	145
Chlorides	0.498	0.248	0.211

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	168	0.239	0.402

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus 26 hours after obstruction. Obstruction released. Neither food nor water postoperatively until 1½ hours after release. Living and apparently healthy 1 week later.

Rabbit 25—Jejunal obstruction—2/9/28. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen...	48.7	111.5
Chlorides	0.487	0.349

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	155	0.193	0.3003

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus 26 hours after obstruction. Obstruction released. Neither food nor water postoperatively until release of obstruction. Living and apparently healthy 1 week later.

Rabbit 26—Jejunal obstruction—2/16/28. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen.....	61.8	128.8	135.3
Chlorides	0.468	0.445	0.376

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	125.5	0.342	0.430

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus 24 hours after obstruction. Obstruction released. Neither food nor water postoperatively until 2 hours after release. Living and apparently healthy 1 week later.

Rabbit 27—Jejunal obstruction—2/16/28. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	3 Hours After Release
Nonprotein nitrogen	55.5	108.5	120
Chlorides	0.505	0.420	0.413

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	153	0.294	0.450

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus 24 hours after obstruction. Obstruction released. Neither food nor water postoperatively until 3 hours after release. Living and apparently well 1 week later.

Rabbit 28—Control—2/16/28. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	At 48 Hours Starvation	At 72 Hours Starvation
Nonprotein nitrogen ..	50	47
Chlorides	0.507	0.498

Normal rabbit starved to determine the effects of special diet and starvation.

Rabbit 29—Jejunal obstruction—3/5/28—Weight 7 pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen	40.8	141	156
Chlorides	0.496	0.380	0.389

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	155	0.256	0.398

Ethylene anesthesia 20 min. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus 24 hours after obstruction. Obstruction released. Neither food nor water postoperatively until 2 hours after release. Rabbit died 20 hours after supposed release of obstruction. There was sufficient necrosis of the bowel wall from too much pressure to cause partial obstruction at point of constriction.

Rabbit 30—Jejunal obstruction—3/12/28. Weight 7½ pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen	39	103.8	86.3
Chlorides	0.481	0.409	0.448

Ethylene anesthesia 18 minutes. Obstruction, rubber band, third inch jejunum. Jejunum accidentally opened below point of obstruction and closed with silk purse-string. There was considerable free fluid in abdominal cavity at time of operation. Blood from ear. Obstruction released in 24 hours. Neither food nor water postoperatively until 2 hours after release of obstruction. Living and apparently healthy 1 week later.

Rabbit 31—Jejunal obstruction—3/12/28. Weight 4½ pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen	52.6	90	104.8
Chlorides	0.502	0.455	0.373

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	120	0.430	0.517

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. Stomach contents removed by catheter through esophagus 24 hours after obstruction. Obstruction released and 150 cc. of distilled water plus 0.4 Gm. NaCl given by catheter through esophagus. Neither food nor water postoperatively until 2 hours after release. Blood from ear. Rabbit died 48 hours after release from partial intestinal obstruction.

Rabbit 32—Jejunal obstruction—3/12/28. Weight 7½ pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release
Nonprotein nitrogen	61.2	102.5	87.8
Chlorides	0.502	0.413	0.426

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	157	0.449	0.705

Ethylene anesthesia 15 min. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Obstruction released and 150 cc. of distilled water plus 0.5 cc. concentrated HCl, the chlorine content of which is equal to 0.4 Gm. NaCl, given by catheter through esophagus. Neither food nor water postoperatively until 2 hours after release. Living and apparently well 1 week later.

Rabbit 33—Jejunal obstruction—3/12/28. Weight 7¼ pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	20 Hours After Release	44 Hours After Release
Nonprotein nitrogen	62.3	160	192	201	217
Chlorides	0.442	0.307	0.297	0.343	0.363

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Obstruction released. Neither food nor water postoperatively until obstruction was released. Animal allowed distilled water ad lib until 20 hours after release, then given food. Died 6 days after obstruction. Slight consolidation of one lung. No other cause of death found.

Rabbit 34—Jejunal obstruction—3/20/28. Weight 5 pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	20 Hours After Release	68 Hours After Release
Nonprotein nitrogen	51.5	126	134	250	63
Chlorides	0.479	0.435	0.403	0.419	0.495

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	101	0.107	0.108

Ethylene anesthesia 15 minutes. Obstruction, third inch jejunum, rubber band. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Obstruction released. Neither food nor water postoperatively until obstruction was released. Animal allowed distilled water ad lib until 20 hours after release, then food and water. Living and apparently healthy 1 week later.

Rabbit 35—Jejunal obstruction—3/26/28. Weight 5 pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	20 Hours After Release	68 Hours After Release
Nonprotein nitrogen	60	129	144	183.5	27
Chlorides	0.462	0.440	0.435	0.452	0.452

Ethylene anesthesia 15 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Attempt to remove stomach contents in 24 hours was unsuccessful. Obstruction released. Neither food nor water postoperatively until stomach was released. Distilled water ad lib until 20 hours after release, then food and water. It is possible that the obstruction in this animal was incomplete which would account for the peculiar figures and also for the unsuccessful attempt to remove stomach contents. Living and apparently healthy 1 week later.

Rabbit 40—Jejunal obstruction—4/9/28. Weight 6 pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	18 Hours After Release	42 Hours After Release
Nonprotein nitrogen.....	51.2	152.6	126.5	156	84
Chlorides	0.528	0.347	0.422	0.336	0.561

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	158	0.395	0.625

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Impossible to empty stomach completely. Obstruction released and 150 cc. of distilled water plus 0.6 Gm. NaCl given by catheter through esophagus. Neither food nor water postoperatively until 2 hours after release, then distilled water ad lib until 18 hours after release, then food and water. Living and apparently healthy 1 week later.

Rabbit 41—Jejunal obstruction. 4/9/28. Weight 7 pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	18 Hours After Release	42 Hours After Release
Nonprotein nitrogen	46	116.2	107	100	169
Chlorides	0.500	0.370	0.439	0.508	0.505

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	215	0.327	0.704

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood froom ear. Stomach contents removed by catheter through esophagus in 24 hours. There was considerable gas in the stomach. Obstruction released. 150 cc. distilled water containing HCl equivalent to 0.6 Gm. NaCl given by catheter through esophagus. Neither food nor water postoperatively until 2 hours after release, then distilled water ad lib until 18 hours after release, then food and water. Rabbit developed pneumonia and died 66 hours after release which probably accounts for the high N.P.N. noted at 42 hours after release.

Rabbit 42—Jejunal obstruction—4/9/28. Weight 5 pounds. Lettuce and bread diet 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	2 Hours After Release	18 Hours After Release	42 Hours After Release
Nonprotein nitrogen	45.8	168	105	100	57.5
Chlorides	0.500	0.307	0.424	0.424	0.508

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach	160	0.328	0.526

Ethylene anesthesia 12 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Obstruction released. 150 cc. distilled water plus 0.6 Gm. NaCl given by catheter through esophagus. Neither food nor water postoperatively until 2 hours after release, then distilled water ad lib until 18 hours after release, then food and water. Living and apparently healthy 1 week later.

Rabbit 61—Jejunal obstruction—5/15/28. Weight 5¼ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ..	55.5	119.1
Chlorides	0.508	0.443
Alkali reserve	.....	82.4

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Bled from heart and killed in 24 hours. Last blood was somewhat clotted. Neither food nor water postoperatively.

Rabbit 62—Jejunal obstruction—5/15/28. Weight 6¾ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ...	40.2	187
Chlorides	0.446	0.194
Alkali reserve	.....	83.7

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Bled and killed in 24 hours. Neither food nor water postoperatively.

Rabbit 70—Ileal obstruction—7/2/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ..	45.1	88.7
Chlorides	0.496	0.415
Alkali reserve	.....	123

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Bled and killed in 24 hours. Almost no distention. Very hot day. Neither food nor water postoperatively.

Rabbit 71—Jejunal obstruction—7/2/28—Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ...	41.3	152.6
Chlorides	0.488	0.420
Alkali reserve	.....	69

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Bled and killed in 24 hours. Slight distention. Very hot day. Neither food nor water postoperatively.

Rabbit 75—Jejunal obstruction—7/9/28. Weight 4½ pounds. Lettuce and bread diet 4 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours Obstruction	30 Hours Obstruction
Nonprotein nitrogen	53	142.8	172.4
Chlorides	0.441	0.404	0.379

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	190	0.604	1.148

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First two blood from ear, third from heart. Bled and killed in 30 hours. Stomach and duodenal contents removed. Rabbit very sick. Neither food nor water postoperatively.

Rabbit 78—Jejunal obstruction—7/23/28. Weight 4½ pounds. Lettuce and bread diet 2 weeks: No food for 48 hours: water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ..	38.9	142
Chlorides	0.488	0.396

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	222	0.334	0.741

Ethylene anesthesia 5 minutes. Obstruction, third inch jejunum, umbilical tape. First blood from ear, second from heart. Bled and killed in 24 hours. Stomach and duodenal contents removed. Rabbit not extremely sick. Neither food nor water postoperatively.

Rabbit No. 79—Ileal obstruction—7/23/28. Weight 4½ pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ..	65.2	104
Chlorides	0.475	0.420

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and intestinal.....	96	0.370	288

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Bled and killed in 24 hours. Stomach and small intestinal contents removed. Rabbit not very sick. Neither food nor water postoperatively.

	Before Obstruction	26 Hours After Obstruction	2 Hours After Release	22 Hours After Release
Nonprotein nitrogen....	49.5	179.3	168.5	127.3
Chlorides	0.518	0.495	0.501	0.508

Rabbit 84—Jejunal obstruction—9/19/29. Lettuce and bread diet 4 weeks: No food for 48 hours.

Ethylene anesthesia 10 minutes. Obstruction, rubber band, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus in 24 hours. Obstruction released. 150 cc. distilled water plus 0.6 Gm. NaCl given by catheter through esophagus. Neither food nor water postoperatively until 22 hours after release of obstruction. Living and apparently healthy 1 week later.

PART II

Rabbit 12—Jejunostomy—3/29/27. General diet: No starvation.

	Before Operation	24 Hours After Operation
Nonprotein nitrogen ...	38	64.1
Chlorides	0.445	0.500

Ethylene anesthesia 20 minutes. Jejunostomy, third inch jejunum, by severing bowel, inserting rubber tube in proximal end and bringing it out through belly wall; distal end tied off and inverted by purse string. Blood from ear. Rabbit died in 36 hours. Jejunostomy functioned fairly well. Food and water allowed throughout procedure.

Rabbit 16—Jejunostomy—11/7/27. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Operation	25½ Hours After Operation
Nonprotein nitrogen ...	43.1	188
Chlorides	0.420	0.317

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	39.5	(which equals no chlorides)	.....

Ethylene anesthesia 20 minutes. Jejunostomy, third inch jejunum, by severing bowel, inserting rubber tube in proximal end and bringing it out through belly wall; distal end tied off and inverted by purse string. Blood from ear. Rabbit killed in 25½ hours. Stomach and duodenal contents removed. Excellent functioning of jejunostomy. Rabbit fairly sick when killed. Neither food no water postoperatively.

Rabbit 17—Jejunostomy—11/7/27. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Operation	25½ Hours After Operation
Nonprotein nitrogen ...	52.1	198
Chlorides	0.450	0.310

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	49.5	(which equals no chlorides)	.....

Ethylene anesthesia 20 minutes. Jejunostomy, third inch jejunum, by severing bowel and suturing wall of proximal end to skin edge; distal end tied off and inverted by purse string. First blood from ear; second from heart. Rabbit killed in 25½ hours. Stomach and duodenal contents removed. Good functioning of jejunostomy. Rabbit fairly sick. Neither food nor water postoperatively.

Rabbit 63—Ileal obstruction—5/15/28. Weight 5 pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ...	39.2	52.2
Chlorides	0.521	0.452
Alkali reserve	.....	74.9

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, terminal ileum. Given 100 cc. water by catheter through esophagus immediately after obstruction. First blood from ear, second from heart. Bled and killed in 24 hours. Last blood slightly clotted. Almost no distention. Rabbit not obviously sick. Neither food nor water postoperatively.

Rabbit 64—Ileal obstruction—5/15/28. Weight 4¾ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ...	48	77.7
Chlorides	0.515	0.368
Alkali reserve	.....	83.7

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 100 cc. water by catheter through esophagus immediately after obstruction. Bled and killed in 24 hours. Rabbit not very sick. Neither food nor water postoperatively.

Rabbit 65—Jejunostomy—6/27/28. Weight 5½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Operation
Nonprotein nitrogen	30.8
Chlorides	0.498

Ethylene anesthesia 20 minutes. Jejunostomy, third inch jejunum, by severing bowel, inserting rubber tube in proximal end and bringing it out through belly wall; distal end tied off and inverted by purse string. Blood from ear. Rabbit died 16 hours after operation because of leak into peritoneal cavity. Allowed water postoperatively.

Rabbit 69—Ileal obstruction—7/2/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	46.8	70
Chlorides	0.500	0.372
Alkali reserve	.....	92

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 100 cc. water by catheter through esophagus immediately after obstruction and the same amount at 2, 4 and 6 hours after obstruction. Bled and killed in 24 hours. Not much distention. Very hot day. Rabbit not especially sick. Neither food nor water postoperatively.

Rabbit 72—Ileal obstruction—7/2/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	57.1	74
Chlorides	0.524	0.417
Alkali reserve	.....	83

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 100 cc. water by catheter through esophagus immediately after obstruction and the same amount 2, 4 and 6 hours after obstruction. Bled and killed in 24 hours. Little distention. Very hot day. Rabbit not especially sick. Neither food nor water postoperatively.

Rabbit 73—Jejunal obstruction—7/9/28. Weight 4½ pounds. Lettuce and bread diet 4 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	30 Hours After Obstruction
Nonprotein nitrogen	51	163.9	222 2
Chlorides	0.425	0.400	0.396

Stomach Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
At 8 hours	66	0.490	0.323
At 16 hours	66	0.366	0.220
At 24 hours	45	0.544	0.245
At 30 hours	10	0.490	0.05

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First two bloods from ear, third from heart. Stomach contents removed by catheter through esophagus at 8, 16 and 24 hours after obstruction. Bled, killed and stomach and duodenal contents removed 30 hours after obstruction. There was no distention of stomach and very little of duodenum. Rabbit had infected jaw. Neither food nor water postoperatively.

Rabbit 74—Jejunal obstruction—7/9/28. Weight 4½ pounds. Lettuce and bread diet 4 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	30 Hours After Obstruction
Nonprotein nitrogen	36.2	109.2	154
Chlorides	0.439	0.401	0.396

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
At 8 hours; stomach	65	0.498	0.324
At 16 hours; stomach	66	0.501	0.331
At 24 hours; stomach	43	0.465	0.200
At 30 hrs.; stomach and duodenal	26	0.340	0.08

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First two bloods from ear, third from heart. Stomach contents removed by catheter through esophagus at 8, 16 and 24 hours after obstruction. Bled, killed and stomach and duodenal contents removed 30 hours after obstruction. No distention of stomach; very slight distention of duodenum. Neither food nor water postoperatively.

Rabbit 76—Jejunal obstruction—7/9/28. Weight 4½ pounds. Lettuce and bread diet 4 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	30 Hours After Obstruction
Nonprotein nitrogen	39.9	129.8	157.3
Chlorides	0.427	0.391	0.349

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
At 30 hrs.; stomach and duodenal	286	0.356	1.018

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First two bloods from ear, third from heart. Given 100 cc. water by catheter through esophagus at 8 hours and again at 16 hours after obstruction. Given 75 cc. water by catheter through esophagus at 24 hours after obstruction. Bled, killed and stomach and duodenal contents removed at 30 hours after obstruction. Rabbit very sick. Considerable distention. Neither food nor water postoperatively.

Rabbit 77—Jejunal obstruction—7/23/28. Weight 4 pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	19 Hours After Obstruction
Nonprotein nitrogen	58.8	200
Chlorides	0.502	0.346

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	188	0.261	0.491

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Given 100 cc. liquid albolene by catheter through esophagus immediately after obstruction and again 7 hours after obstruction. Dying, bled and killed 19 hours after obstruction. Stomach and duodenal contents removed. 200 cc. albolene discarded, the weight of stomach and duodenal contents being that left after removal of albolene. Neither food nor water postoperatively.

Rabbit 80—Ileal obstruction—7/23/28. Weight 4½ pounds. Lettuce and bread diet 2 weeks: No food for 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	43.3
Chlorides	0.492

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and intestinal	160	0.286	0.458

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. Blood from ear. Given 100 cc. liquid albolene by catheter through esophagus immediately after obstruction and again 7 hours later. Given 75 cc. glycerin by catheter through esophagus 11 hours after obstruction. The glycerin was given by mistake instead of albolene. Rabbit died during night. Considerable hemorrhage into stomach. Stomach and duodenal contents removed a few hours after death. Neither food nor water postoperatively.

Rabbit 85—Jejunal obstruction—9/20/28. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22 Hours After Obstruction
Nonprotein nitrogen	53.7	122
Chlorides	0.467	0.430

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Given 120 cc. liquid albolene by catheter through esophagus immediately after obstruction. Bled and killed 22 hours after obstruction. Fairly sick. Neither food nor water postoperatively.

Rabbit 86—Jejunal obstruction—9/20/28. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22 Hours After Obstruction
Nonprotein nitrogen	49.6	177
Chlorides	0.512	0.429

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Given 120 cc. of stomach contents (obtained from rabbit 83 by catheter through esophagus 24 hours after jejunal obstruction) by catheter through esophagus immediately after obstruction. Stomach contents used within 1 hour after removal from rabbit 83. Bled and killed 22 hours after obstruction. Rabbit fairly sick. Neither food nor water postoperatively.

Rabbit 87—Jejunal obstruction—9/20/28. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22 Hours After Obstruction
Nonprotein nitrogen ...	60	203
Chlorides	0.508	0.430

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Given 120 cc. of stomach contents (obtained from rabbit 84 by catheter through esophagus 24 hours after jejunal obstruction) by catheter through esophagus immediately after obstruction. Stomach contents used within 1 hour after removal from rabbit 84. Bled and killed 22 hours after obstruction. Rabbit fairly sick. Neither food nor water postoperatively.

Rabbit 88—Ileal obstruction—9/25/28. Weight 5 pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	36.6
Chlorides	0.492

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. Blood from ear. Given 180 cc. liquid albolene by catheter through esophagus immediately after obstruction. Given 100 cc. liquid albolene by catheter through esophagus 8 hours after obstruction. Rabbit died during night; greatly distended, oil filling entire small intestine. Neither food nor water postoperatively.

Rabbit 89—Ileal obstruction—9/25/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22½ Hours After Obstruction
Nonprotein nitrogen ...	48.3	166.6
Chlorides	0.508	0.369

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 180 cc. liquid albolene by catheter through esophagus immediately after obstruction. Given 100 cc. liquid albolene by catheter through esophagus 8 hours after obstruction. Dying, bled and killed 22½ hours after obstruction. Enormous distention. Neither food nor water postoperatively.

Rabbit 90—Ileal obstruction—9/25/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22½ Hours After Obstruction
Nonprotein nitrogen ...	46.1	87.3
Chlorides	0.495	0.475

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 180 cc. stomach and duodenal contents (obtained from rabbit obstructed at third inch jejunum for 22 hr.) by catheter through esophagus immediately after obstruction. Given 100 cc. stomach and duodenal contents (obtained from rabbit obstructed at third inch jejunum for 26 hours) by catheter through esophagus 6 hours after obstruction. The stomach and duodenal contents were obtained from rabbits obstructed the previous day, killed at the end of 22 and 26 hours respectively, and their stomach and duodenal contents removed. The contents were used for ingestion into rabbit 90 within 1 hour after the death of the respective rabbit. Bled and killed 22½ hours after obstruction. Fairly sick but not nearly so much distention as was noted in 88, 89, 91. Neither food nor water postoperatively.

Rabbit 91—Ileal obstruction—9/25/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22½ Hours After Obstruction
Nonprotein nitrogen ...	50.1	157.7
Chlorides	0.512	0.438

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 180 cc. liquid albolene by catheter through esophagus immediately after obstruction. Given 100 cc. liquid albolene by catheter through esophagus 8 hours after obstruction. Bled and killed 22½ hours after obstruction. Rabbit very sick. Greatly distended. Neither food nor water postoperatively.

Rabbit 92—Jejunal obstruction—9/27/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen ...	46.1	152.6
Chlorides	0.515	0.469

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, third inch jejunum. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. Blood from ear. Died between 32 and 34 hours after operation. Stomach drained well so that there was no distention of stomach and very little of duodenum. Very little peritoneal reaction. Water ad lib postoperatively. No food.

Rabbit 94—Control operation—10/9/28. Weight 5 pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	46 Hours After Obstruction
Nonprotein nitrogen	40.5	58	48.7
Chlorides	0.462	0.462	0.479

Ethylene anesthesia 10 minutes. Anterior stomach wall sutured to anterior abdominal wall. Blood from ear. Water ad lib postoperatively; food after 46 hours. Rabbit living and apparently healthy 1 week later.

Rabbit 95—Pyloric obstruction—10/9/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	28 Hours After Obstruction
Nonprotein nitrogen	46.1	202	232.5
Chlorides	0.446	0.347	0.330

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. There was considerable bleeding from incised stomach which was finally controlled by many sutures. First two bloods from ear, third from heart. Dying, bled and killed 28 hours after operation. There was a slight amount of hemorrhage around the pylorus and a small hematoma of the stomach wall. Very little peritoneal irritation. Water ad lib postoperatively. No food.

Rabbit 93—Pyloric obstruction—10/9/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	44 Hours After Obstruction
Nonprotein nitrogen	50	103.6	243.9
Chlorides	0.420	0.366	0.257

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening through a wide incision and suturing mucous membrane to skin. First two bloods from ear, third from heart. Dying, bled and killed 44 hours after operation. Very little peritoneal irritation. Water ad lib postoperatively. No food.

Rabbit 97—Control operation—10/9/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Operation	24 Hours After Operation	46 Hours After Operation
Nonprotein nitrogen	40	60	54.9
Chlorides	0.478	0.470	0.479

Ethylene anesthesia 10 minutes. Anterior stomach wall sutured to anterior abdominal wall. Blood from ear. Water ad lib postoperatively. Food after 46 hours. Living and apparently healthy 1 week later.

Rabbit 102—Jejunal obstruction—11/1/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	56.8
Chlorides	0.482

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, third inch jejunum. Given 120 cc. 0.9 per cent NaCl by catheter through esophagus immediately after obstruction. Blood from ear. Died during night from ruptured duodenum. Neither food nor water postoperatively.

Rabbit 103—Ileal obstruction—11/1/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	22½ Hours After Obstruction
Nonprotein nitrogen ...	46	116
Chlorides	0.475	0.424

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, terminal ileum. First blood from ear, second from heart. Given 180 cc. stomach and duodenal contents (obtained from rabbit with jejunal obstruction at 26 hours) by catheter through esophagus immediately after obstruction. Given 100 cc. stomach and duodenal contents (obtained from rabbit with jejunal obstruction at 26 hours) by catheter through esophagus 5 hours after obstruction. The stomach and duodenal contents were obtained from rabbits obstructed at third inch of jejunum the previous day and were used for rabbit 103 within 1 hour after the death of the rabbits. Bled and killed 22½ hours after obstruction. Neither food nor water postoperatively. Not very sick.

Rabbit 104—Jejunal obstruction—11/1/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	55.2
Chlorides	0.492

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, third inch jejunum. Given 120 cc. stomach and duodenal contents (obtained from rabbit with jejunal obstruction for 26 hours) by catheter through esophagus immediately after obstruction. Stomach and duodenal contents used were obtained from rabbit obstructed the previous day and were used within 1 hour, after the death of the rabbit. Blood from ear. Died during night. Neither food nor water postoperatively.

Rabbit 105—Jejunal obstruction—11/1/28. Weight 4½ pounds. General diet: No food for 48 hours; water ad lib.

	Before Obstruction	20 Hours After Obstruction
Nonprotein nitrogen	51	195
Chlorides	0.515	0.386

Ethylene anesthesia 5 minutes. Obstruction, umbilical tape, third inch jejunum. Given 120 cc. liquid albolene by catheter through esophagus immediately after obstruction. First blood from ear. Second from heart. Dying, bled and killed in 20 hours. Neither food nor water postoperatively.

PART III

Rabitt 36—Jejunal obstruction—4/2/28. Weight 4¾ pounds. Lettuce and bread diet for 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	40	104
Chlorides	0.491	0.479

Stomach Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
At 8 hours	134.5	0.410	0.552
At 16 hours	149.0	0.225	0.336
At 24 hours	84.0	0.390	0.328
At 32 hours	64.0	0.267	0.171

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, third inch jejunum. Blood from ear. Stomach contents removed by catheter through esophagus at 8, 16, 24 and 32 hours after obstruction. 40 cc. 0.9 per cent NaCl given intravenously at 8, 16, 24 and 32 hours after obstruction. Rabbit died between 37 and 39 hours after obstruction. The duodenum was greatly distended, but the stomach was approximately normal in size and appearance. Neither food nor water postoperatively.

Rabbit 37—Jejunal obstruction—4/2/28. Weight 3½ pounds. Lettuce and bread diet for 3 weeks: No food for 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction
Nonprotein nitrogen	49.5	112.5
Chlorides	0.473	0.528

Stomach Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
At 8 hours	128	0.343	0.439
At 16 hours	99	0.067	0.066

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. Stomach contents removed by catheter through esophagus at 8 and 16 hours after obstruction. Dying, bled and killed 24 hours after obstruction. Duodenum greatly distended. Stomach normal in size and appearance. Neither food nor water postoperatively.

Rabbit 38—Jejunal obstruction—4/2/28. Weight 4½ pounds. Lettuce and bread diet 2 weeks: No food 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	29 Hours After Obstruction
Nonprotein nitrogen	35.8	85	111
Chlorides	0.531	0.554	0.620

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	290	0.329	0.951

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, third inch jejunum. First two bloods from ear, third from heart. 40 cc. 0.9 per cent NaCl given intravenously at 8, 16 and 24 hours after obstruction. Bled and killed 29 hours after obstruction. Fairly sick. Stomach and duodenum greatly distended. Neither food nor water postoperatively.

Rabbit 39—Jejunal obstruction—4/2/28. Weight 4½ pounds. Lettuce and bread diet 2 weeks: No food 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	29 Hours After Obstruction
Nonprotein nitrogen	38.3	118.5	143
Chlorides	0.538	0.502	0.634

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	390	0.353	1.377

Ethylene anesthesia 8 minutes. Obstruction, umbilical tape, third inch jejunum. First two bloods from ear, third from heart. 40 cc. NaCl given intravenously at 8, 16 and 24 hours after obstruction. Bled and killed 29 hours after obstruction. Fairly sick. Stomach and duodenum greatly distended. Neither food nor water postoperatively.

Rabbit 43—Jejunostomy—4/16/28. Lettuce and bread diet 10 days: No food 48 hours; water ad lib.

	Before Operation	24 Hours After Operation
Nonprotein nitrogen	53	96.6
Chlorides	0.496	0.545

Ethyene anesthesia 10 minutes. Jejunostomy, third inch jejunum, by bringing loop out through skin, completely severing it and tying off distal end. Blood from ear. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after operation. Rabbit died 36 hours after operation. There was poor functioning of jejunostomy. Stomach and duodenum greatly distended. Neither food nor water postoperatively.

Rabbit 44—Jejunostomy—4/16/28. Lettuce and bread diet 10 days: No food 48 hours; water ad lib.

	Before Operation	24 Hours After Operation	37 Hours After Operation
Nonprotein nitrogen	58.8	148.8	274
Chlorides	0.512	0.429	0.375

Ethylene anesthesia 10 minutes. Jejunostomy, third inch jejunum, by bringing loop out through skin, completely severing it and tying off distal end. First two bloods from ear, third from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after operation. Bled and killed 37 hours after operation. Fairly sick. Fair functioning of jejunostomy although there was some distention of stomach and duodenum. Neither food nor water postoperatively.

Rabbit 45—Jejunostomy—4/16/28. Lettuce and bread diet 10 days: No food 48 hours; water ad lib.

	Before Operation	24 Hours After Operation	37 Hours After Operation
Nonprotein nitrogen	60	151.5	251
Chlorides	0.462	0.440	0.390

Ethylene anesthesia 10 minutes. Jejunostomy, third inch jejunum, by bringing out loop through skin, completely severing it and tying off distal end. First two bloods from ear, third from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after operation. Bled and killed 37 hours after operation. Quite sick. Poor functioning of jejunostomy since duodenum was considerably distended. Neither food nor water postoperatively.

Rabbit 46—Jejunal obstruction—4/23/28. Weight 4½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen	38	125.5
Chlorides	0.475	0.353

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	278	0.318	0.884

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after obstruction. Bled and killed in 26 hours. Fairly sick. Stomach and duodenal contents removed. Neither food nor water postoperatively.

Rabbit 47—Jejunal Obstruction—4/23/28. Weight 3¾ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen	53	124.5
Chlorides	0.473	0.538

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal	251	0.431	1.082

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after obstruction. Bled and killed in 26 hours. Fairly sick. Stomach and duodenal contents removed. Neither food nor water postoperatively.

Rabbit 48—Jejunal obstruction—4/23/28. Weight 3½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen ...	51	139.5
Chlorides	0.472	0.340

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	217	0.389	0.846

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after obstruction. Bled and killed in 26 hours. Fairly sick. Stomach and duodenal contents removed. Neither food nor water postoperatively.

Rabbit 49—Jejunal obstruction—4/23/28. Weight 3¼ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen ...	56	140.3
Chlorides	0.515	0.545

Contents	Weight, Gm.	Per Cent as NaCl	Total as NaCl, Gm.
Stomach and duodenal.....	246	0.388	0.955

Ethylene anesthesia 6 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after obstruction. Bled and killed in 26 hours. Fairly sick. Stomach and duodenal contents removed. Neither food nor water postoperatively.

Rabbit 50—Control—4/27/28. Weight 2½ pounds. General diet: No starvation.

	Before	24 Hours After
Nonprotein nitrogen ...	33.5	62.8
Chlorides	0.462	0.412
Alkali reserve	.....	95

First blood from ear, second from heart. 20 cc. 3 per cent sodium bicarbonate given intravenously at the beginning and again in 12 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib.

Rabbit 51—Control—4/27/28. Weight 2½ pounds. General diet: No starvation.

	Before	24 Hours After
Nonprotein nitrogen ...	30.5	56.5
Chlorides	0.459	0.402
Alkali reserve	.....	86.2

First blood from ear, second from heart. 20 cc. 3 per cent sodium bicarbonate given intravenously at the beginning and again in 12 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib.

Rabbit 52—Control—5/4/28. Weight 5½ pounds. General diet: No food 48 hours; water ad lib.

	Before	24 Hours After
Nonprotein nitrogen ...	37	41
Chlorides	0.492	0.517
Alkali reserve	.....	61.4

First blood from ear, second from heart. 30 cc. 2½ per cent dextrose given intravenously at the beginning and again in 10 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib.

Rabbit 53—Control—5/4/28. Weight 4¾ pounds. General diet: No food 48 hours; water ad lib.

	Before	24 Hours After
Nonprotein nitrogen ...	32	42
Chlorides	0.479	0.462
Alkali reserve	.....	65.4

First blood from ear, second from heart. 30 cc. 3 per cent sodium bicarbonate neutralized with lactic acid given intravenously at the beginning and again in 10 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib.

Rabbit 54—Control—5/4/28. Weight 5½ pounds. General diet: No food 48 hours; water ad lib.

	Before	24 Hours After
Nonprotein nitrogen ...	35.7	46.5
Chlorides	0.492	0.465
Alkali reserve	.....	54

First blood from ear, second from heart. 20 cc. 1 per cent lactic acid given intravenously at beginning and again in 10 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib. Attempts to give 3 per cent lactic acid intravenously killed 3 rabbits immediately.

Rabbit 55—Control—5/4/28. Weight 5 pounds. General diet: No food 48 hours; water ad lib.

	Before	24 Hours After
Nonprotein nitrogen ...	37.2	61.2
Chlorides	0.498	0.469
Alkali reserve	.....	81.5

First blood from ear, second from heart. 30 cc. 3 per cent sodium bicarbonate given intravenously at the beginning and again in 10 hours. Bled and killed 24 hours after first injection. No food after first injection. Distilled water ad lib.

Rabbit 56—Control—5/15/28. Weight 5 pounds. General diet: No food 24 hours; water ad lib. Alkali reserve.....56.4. Blood from heart; rabbit killed.

Rabbit 57—Jejunal obstruction—5/7/28. Weight 5½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	39.3
Chlorides	0.479

Ethylene anesthesia 7 minutes. Obstruction, umbilical tape, third inch jejunum. Blood from ear. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after obstruction. Died about 18 hours after obstruction. Stomach and duodenum enormously distended. Neither food nor water postoperatively.

Rabbit 58—Jejunal obstruction—5/7/28. Weight 5½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	20 Hours After Obstruction
Nonprotein nitrogen ...	42.5	152.6
Chlorides	0.528	0.380
Alkali Reserve	.....	65.5

Ethylene anesthesia 7 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after obstruction. Bled and killed 20 hours after obstruction. Not very sick. Neither food nor water postoperatively.

Rabbit 59—Jejunal obstruction—5/7/28. Weight 6 pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	20 Hours After Obstruction
Nonprotein nitrogen ...	48.7	132.4
Chlorides	0.503	0.538
Alkali reserve	.....	58

Ethylene anesthesia 7 minutes. Obstruction, umbilical tape, third inch jejunum. First blood froom ear, second from heart. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after obstruction. Bled and killed 20 hours after obstruction. Not very sick. Neither food nor water postoperatively.

Rabbit 60—Jejunal obstruction—5/7/28. Weight 5¼ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	20 Hours After Obstruction
Nonprotein nitrogen ...	36.3	103.6
Chlorides	0.515	0.373
Alkali reserve	.....	63.5

Ethylene anesthesia 7 minutes. Obstruction, umbilical tape, third inch jejunum. First blood from ear, second from heart, 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after obstruction. Bled and killed 20 hours after obstruction. Not very sick. Neither food nor water postoperatively.

Rabbit 66—Jejunostomy—6/27/28—Weight 4½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	26 Hours After Obstruction
Nonprotein nitrogen ...	75.9	86.6
Chlorides	0.465	0.501

Ethylene anesthesia 15 minutes. Jejunostomy, third inch jejunum, by severing jejunum, placing small rubber tube in proximal end and bringing it out through belly wall. Distal end tied off and inverted by purse string. First blood from ear, second from heart. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after operation. Bled and killed in 26 hours. Very sick. Very slight peritoneal irritation. Stomach almost empty and but little dilation of duodenum. Neither food nor water postoperatively.

Rabbit 67—Jejunostomy—6/27/28. Weight 5 pounds. General diet: No food 48 hours; water ad lib.

	Before Operation	26 Hours After Operation
Nonprotein nitrogen ...	39.8	107
Chlorides	0.498	0.460

Ethylene anesthesia 15 minutes. Jejunostomy, third inch jejunum, by severing jejunum, placing rubber tube in proximal end and bringing it out through belly wall. Distal end tied off and inverted by purse string. First blood from ear, second from heart. 40 cc. 5 per cent dextrose given intravenously 8 and 16 hours after operation. Bled and killed 26 hours after operation. Quite sick. Slight peritoneal irritation. Stomach fairly well emptied. Duodenum slightly distended. Neither food nor water postoperatively.

Rabbit 68—Jejunostomy—6/27/28. Weight 4½ pounds. General diet: No food 48 hours; water ad lib.

	Before Operation	26 Hours After Operation
Nonprotein nitrogen ...	50	113
Chlorides	0.498	0.564

Ethylene anesthesia 15 minutes. Jejunostomy, third inch jejunum, by severing jejunum, placing rubber tube in proximal end and bringing it out through belly wall. Distal end tied off and inverted by purse string. First blood from ear, second from heart. 40 cc. 0.9 per cent NaCl given intravenously 8 and 16 hours after operation. Very sick. Bled and killed in 26 hours. Slight peritoneal irritation. Stomach nearly empty. Duodenum slightly distended. Neither food nor water postoperatively.

Rabbit 82—Jejunostomy—9/17/28. General diet: No food 48 hours; water ad lib.

	Before Operation	24 Hours After Operation	47 Hours After Operation
Nonprotein nitrogen	58.8	112.3	224.3
Chlorides	0.508	0.578	0.548

On 7/16/28 preliminary operation was done bringing loop of jejunum up through abdominal wall and suturing it in position, leaving only skin covering intestine; ½ per cent procaine hydrochloride anesthesia. Skin opened. Jejunum severed, distal end tied off. First two bloods from ear, third from heart. 40 cc. 0.9 per cent NaCl given intravenously 8, 16, 24, 32 and 40 hours after operation. Dying, bled and killed in 47 hours. Stomach empty. Very slight dilation of duodenum. Neither food nor water postoperatively.

Rabbit 93—Jejunal obstruction—9/27/28. Weight 4½ pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	24 Hours After Obstruction	36 Hours After Obstruction
Nonprotein nitrogen	44	157.4	208.3
Chlorides	0.459	0.452	0.376

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, third inch jejunum. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. First two bloods from ear, third from heart. 40 cc. 2½ per cent dextrose given subcutaneously 8, 16, 24 and 32 hours after obstruction. Dying, bled and killed in 36 hours. Very little peritoneal irritation. Stomach empty. Duodenum moderately distended. Neither food nor water postoperatively.

Rabbit 98—Pyloric obstruction—10/17/28. Weight 4 pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	28 Hours After Obstruction
Nonprotein nitrogen ...	46.8	240.9
Chlorides	0.462	0.307

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. First blood from ear, second from heart. 40 cc. 2½ per cent dextrose given intravenously 7, 13 and 23 hours after obstruction. Dying, bled and killed in 28 hours. Very slight peritoneal irritation. Stomach empty. No food. Water ad lib.

Rabbit 99—Pyloric obstruction—10/17/28. Weight 4 pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	44.7
Chlorides	0.495

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. Blood from ear. Considerable bleeding from stomach wall controlled by many sutures. 40 cc. 0.9 per cent NaCl given intravenously 7 and 13 hours after obstruction. Died during night. No food. Water ad lib.

Rabbit 100—Pyloric obstruction—10/17/28. Weight 4 pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction
Nonprotein nitrogen	39.7
Chlorides	0.476

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening stomach through a wide incision and suturing mucous membrane to skin. Moderate bleeding from stomach. Blood from ear. 40 cc. 2½ per cent dextrose given intravenously 7 and 13 hours after obstruction. Died during night. No food. Water ad lib.

Rabbit 101—Pyloric obstruction—10/17/28. Weight 4 pounds. General diet: No food 48 hours; water ad lib.

	Before Obstruction	48 Hours After Obstruction	67 Hours After Obstruction
Nonprotein nitrogen	45.4	145.9	229.8
Chlorides	0.488	0.393	0.343

Ethylene anesthesia 15 minutes. Obstruction, umbilical tape, pylorus. Gastrostomy by opening stomach through wide incision and suturing mucous membrane to skin. First two bloods from ear, third from heart. 40 cc. 0.9 per cent NaCl given intravenously 7 and 13 hours after operation and subcutaneously 30, 35, 46, 52 and 58 hours after operation. Unable to bleed at 24 hours. Bled with difficulty at 48 hours. Dying, bled and killed at 67 hours. Stomach empty. Very slight peritoneal irritation. No food. Water ad lib.

A COMPARATIVE STUDY OF THE BACTERICIDAL VALUES OF TWENTY-ONE COMMONLY USED ANTISEPTICS *

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NEW YORK

This paper is the third in a series of studies of antiseptics. The first was a study of the bactericidal effect of mercurochrome-220 soluble [1] on *Bacillus pyocyaneus,* while the second [2] was an investigation of some of the properties of the antiseptic known as S. T. 37, described in 1927 by Leonard and Feirer.[3] The present article is the direct outgrowth of the first two papers, and has for its object a comparative study, in vitro, of the bactericidal effect of twenty-one commonly used antiseptics on four different organisms growing on agar slants.

The organisms used were a freshly isolated strain of *Staphylococcus aureus,* a similar strain of *Bacillus pyocyaneus,* and laboratory stock cultures of *Bacillus subtilis* and *Bacillus typhosus.* Preliminary to the actual experimental work, each culture was transplanted for five successive days on agar slants. Immediately preceding experimentation, two fresh transplants were made from the fifth daily culture to agar slants. These were labeled B and X, while the parent culture was labeled A. Culture X served as a control of growth; A was a twenty-four hour old culture; while B was a freshly inoculated slant. Twenty-one such groups were made up using each of the four organisms employed (fig. 1).

To each of the twenty-one A and B groups of the four cultures, a different antiseptic was added in such quantity that when the culture tube was held in the vertical position, the slant surface was entirely covered by the antiseptic solution, insuring continuous contact of the agar slant surface with its culture and the antiseptic. At intervals of 15, 30, 60 and 180 minutes, transplants were made from the A and B tubes to fresh agar slants A′, A″, B′, B″, etc. A previously flamed

* Submitted for publication, March 30, 1929.

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1. Wright, I. S.: Effect of Mercurochrome-220 Soluble on the Growth of Bacillus Pyocyaneus, Arch. Surg. **15**:958 (Dec.) 1928.

2. Allen, A. W., and Wright, I. S.: The Bactericidal Properties of the Solution S. T. 37, Arch. Surg. **17**:834 (Nov.) 1928.

3. Leonard, Veader; and Feirer, W. A.: Bactericidal Activity of Hexylresorcinol in Glycerine, Bull. Johns Hopkins Hosp. **41**:21 (May) 1927; Hexylresorcinol as a General Antiseptic, Surg. Gynec. Obst. **45**:603 (Nov.) 1927.

platinum loop was inserted into the supernatant antiseptic fluid, down to the agar surface, where the loop was drawn across the agar slant surface with its bacterial covering. This loopful of suspension was withdrawn through the antiseptic fluid above and streaked on a fresh agar slant. These tubes plus the originals were incubated at 37 C. for twenty-four hours and then observed. The entire experiment was repeated as a check on the first series.

The antiseptics used were obtained from the hospital pharmacy and conformed to the requirements as indicated in table 1. Dakin's solution was prepared according to the directions of Carrel,[4] and used the same day. Both Dakin's solution and Zonite were titrated by the method of the same author. The agar was the usual meat infusion agar titrated to a p_H of 7.4.

Of the antiseptics listed, some are used routinely preoperatively, or as prophylactics against infection; others are applied to grossly

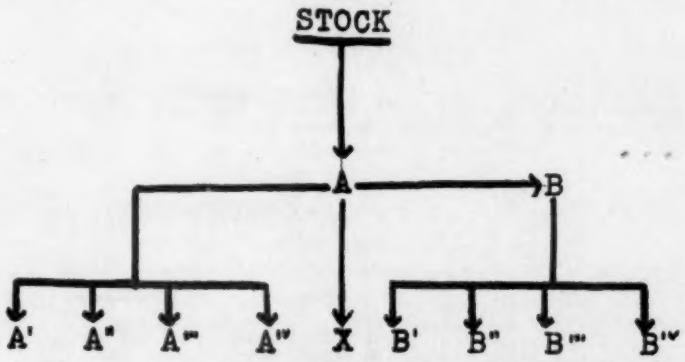

Fig. 1.—A diagram showing the origin of the cultures.

infected wounds; those of a third group are employed as antiseptic or germicidal irrigants. In this discussion, no such classification is made. Instead, they are arbitrarily grouped as dyes, halogens, heavy metals, miscellaneous, oxidizers and phenols, according to their exact or most apparent composition (table 2). Obviously, such a classification is approximate at best, and open to criticism. However, this classification with its shortcomings serves the purpose of this paper. It may be remarked in passing that the miscellaneous group should perhaps be called the alcoholic group, since all but one contain a large percentage of alcohol in addition to the other substances present.

This method of determining the bactericidal values of antiseptics is open to criticism. It may be said that it is not a standard method of determining the bactericidal properties of antiseptics. In practice, one

4. Carrel, A.: Communication to the Editor, J. A. M. A. **67**:1777 (Dec.) 1916.

does not routinely or continuously immerse the contaminated or infected field in any antiseptic. Still less is one able to suspend the organisms in the antiseptic as represented by the Rideal-Walker [5] or the Hygienic Laboratory procedure.[6]

In this work, attention is called to the fact that the penetrating power of each antiseptic was allowed to exert whatever influence it

TABLE 1.—*The Antiseptics Used in the Study, with Notes*

Dichloramine T, 5 per cent N.N.R.
Harrington's solution (Harrington, Charles: Some Studies in Asepsis, Ann. Surg. **40**: 475 [Oct.] 1904)
Silver nitrate, 10 per cent, prepared and used the same day
Zonite, original bottle, full strength, titer 1.10 per cent NaClO
Cresol compound, 2 per cent, U.S.P. (lysol)
Potassium mercuric iodide of the salt, Gm. 4, acetone 460 cc.
Dakin's solution, N.N.R., prepared and used the same day, titer 0.5 per cent NaCl
Tincture of green soap, U.S.P.
Solution of iodine (Lugol's solution) 5 per cent, U.S.P.
Tincture of iodine, 7 per cent, U.S.P.
Protargin mite (argyrol) 20 per cent, N.N.R., prepared and used the same day
Alcohol, ethyl, 80 per cent
Hydrogen peroxide, solution, U.S.P., original recently received bottle
S. T. 37 (solution hexylresorcinolis 1 : 1,000), original bottle
Mercuric chloride solution, 1 : 1,000
Mercurochrome-220 soluble, 2 per cent aqueous solution, N.N.R.
Listerine, original bottle, full strength
Phenol, 1 : 1,000
Acriflavine, neutral, 1 : 1,000, N.N.R., prepared and used the same day
Potassium permanganate, 1 : 1,000, prepared and used the same day
Boric acid, saturated solution at room temperature

TABLE 2.—*The Classification of the Antiseptics Employed, with the Number of Sterile Transplants for Each Antiseptic*

Halogens		**Miscellaneous**	
Dichloramine T, 5 per cent	64	Harrington's solution	64
Zonite	61	Potassium mercuric iodide	50
Dakin's solution	49	Tincture of green soap	49
Solution of iodine, 5 per cent	48	Alcohol, ethyl, 80 per cent	42
Tincture of iodine, 7 per cent	48	Listerine	13
Heavy Metals		**Phenols**	
Silver nitrate, 10 per cent	64	Cresol compound (lysol) 2 per cent	51
Protargin mite (argyrol)	42	S. T. 37, sol. hexylresorcinal, 1:1,000	21
Mercuric chloride, 1:1,000	18	Phenol, 1:1,000	10
Boric acid	3		
Oxidizers		**Dyes**	
Hydrogen peroxide	38	Mercurochrome, 2 per cent	16
Potassium permanganate	7	Acriflavine, neutral, 1:1,000	8

possessed on the bacteria; that incident to transplanting the bacteria there was a certain degree of agitation of the bacteria causing a certain amount of suspension of the organisms in the antiseptic solution for a shorter period of time than that named; and that, in addition, a certain

5. Rideal, S., and Walker, J. T. A.: Standardization of Disinfectants, J. Roy. San. Inst. **24**:424, 1903.

6. Anderson, J. F., and McClintock, T. B.: A Method for the Bacteriological Standardization of Antiseptics, J. Infect. Dis. **8**:1, 1911. Harrington, Charles: Some Studies in Asepsis, Ann. Surg. **40**:475 (Oct.) 1904.

amount of the antiseptic was of necessity carried over to the fresh agar slants along with each inoculum of the A′, B′, etc., tubes.

In practice, one deals with two situations; the one a massive infection characterized usually by pus; the other, mere contamination. The present schema was devised to compare simultaneously, and under as nearly exact conditions as possible, the germicidal effect of the twenty-one antiseptics, under stated conditions, (1) on the type of organism used in determining the phenol coefficient; (2) on three different organ-

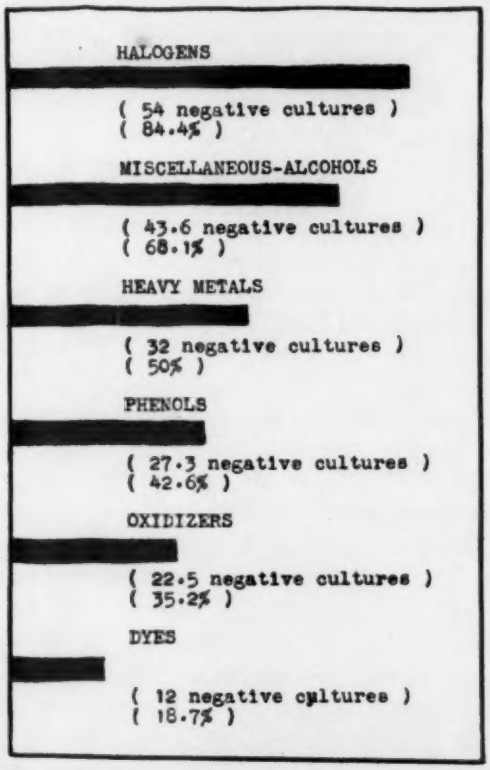

Fig. 2.—A diagram showing the relative efficiency of each of the six types of antiseptics used.

isms, roughly typical of three different classes of invaders; (3) under a condition simulating that most frequently met in routine practice: namely, surface and subsurface massive contamination.

Bacillus typhosus was employed because it is the organism used at present in determining the phenol coefficient of antiseptics. *Staphylococcus aureus* is a common contaminating and infecting organism relatively resistant to antiseptics in vivo, and is roughly characteristic of the pyogenic group. *Bacillus pyocyaneus* is a relatively frequent secondary invader. It is generally more resistant to antiseptics than

TABLE 3.—*The Antiseptics Used, Ranked in Order of Bactericidal Power*

Antiseptic	Subcultures *																																	
	Staphylococcus Aureus									Bacillus Pyocyaneus									Bacillus Subtilis									Bacillus Typhosus								
	A				B				X	A				B				X	A				B				X	A				B				X
	1	2	3	4	1	2	3	4		1	2	3	4	1	2	3	4		1	2	3	4	1	2	3	4		1	2	3	4	1	2	3	4	
Dichloramine T	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+
Harrington's solution	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+
Silver nitrate	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+
Zonite	+	−	−	−	−	−	−	−	+	+	+	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+
Cresol compound	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+
Potassium mercuric iodide	+	+	−	−	−	−	−	−	+	+	+	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	+
Dakin's solution	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	+	−	−	−	+	−	−	−	+	−	−	−	−	−	−	−	−	+
Tincture of green soap	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	+	+	+	−	+	+	+	−	+	−	−	−	−	−	−	−	−	+
Solution of iodine	+	+	+	+	+	+	+	−	+	+	−	−	−	−	−	−	−	+	+	−	−	−	+	−	−	−	+	+	−	−	−	−	−	−	−	+
Tincture of iodine	−	−	−	−	−	−	−	−	+	+	+	−	−	−	−	−	−	+	+	+	+	−	+	+	+	−	+	−	−	−	−	−	−	−	−	+
Protargin mite	+	+	−	−	−	−	−	−	+	+	−	−	−	−	−	−	−	+	+	+	+	−	+	+	+	−	+	−	−	−	−	−	−	−	−	+
Alcohol	−	−	−	−	−	−	−	−	+	−	−	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	+
Hydrogen peroxide	−	−	+	−	+	−	−	−	+	+	−	−	−	+	−	−	−	+	+	+	+	−	+	+	+	−	+	−	−	−	−	−	−	−	−	+
S. T. 37	+	+	−	−	+	−	−	−	+	+	+	−	−	−	−	−	−	+	+	+	+	−	+	+	+	−	+	−	−	−	−	−	−	−	−	+
Mercuric chloride	+	+	+	+	−	−	−	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	+
Mercurochrome	+	+	+	+	+	+	−	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	+
Listerine	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	+
Phenol	+	+	+	−	+	+	−	−	+	+	+	+	−	+	+	+	−	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	−	−	−	+
Acriflavine	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Potassium permanganate	+	+	−	−	+	−	−	−	+	+	+	+	+	+	+	+	−	+	+	+	+	+	+	+	+	+	+	+	+	−	−	−	+	−	−	+
Boric acid	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+

* + indicates growth of transplant; −, no growth of transplant.

Staphylococcus aureus. It is somewhat typical of the gram-negative bacilli. *Bacillus subtilis,* while not a pathogen, is a spore former, and was used because of that characteristic.

Omitting the control cultures, there were sixty-four transplants of each organism-antiseptic group. Since the negative cultures are indicative of germicidal value, the antiseptics have been listed in descending order of the number of sterile subcultures (table 3). In computing the results for figure 2, the negative cultures of each antiseptic of the six major subdivisions have been totaled and divided by the component number of the group. The percentages expressed are on the basis of sixty-four. From this computation, and also table 3, it is evident that the comparative rating of the antiseptic groups is as follows: halogens, miscellaneous, heavy metals, phenols, oxidizers and dyes.

COMMENT

The value of an antiseptic is commonly expressed in terms of its phenol coefficient. Especially is this true commercially. The bacterium employed in the determination of the phenol coefficient is the *Bacillus typhosus.* Although this procedure is of definite theoretic value, from a practical point of view, it has certain disadvantages. It is known that the organism of typhoid fever has relatively little resistance against many of the commonly used antiseptics. This is well illustrated by the results shown in table 3. If one contrasts the number of positive transplants of the typhoid group with the number of similar subcultures of any other group, it is obvious that the phenol coefficient is at best but superficially relevant to the actual practical bactericidal value of any antiseptic. It is a remote approximation.

CONCLUSIONS

1. The bactericidal efficacy of each of twenty-one commonly used antiseptics was tested against four different organisms.

2. The halogen group, as determined by this study, is the most efficacious of the antiseptic groups.

3. The phenol coefficient value stated as an index of practical bactericidal efficacy is unsatisfactory. The present methods of judging antiseptics do not constitute an accurate index of their practical worth.

4. The need for a comparative study of the bactericidal value of an antiseptic under a variety of conditions before it is offered to the public will be discussed in a later communication.

SURGICAL DISEASES OF THE COLON

COOPERATIVE MANAGEMENT *

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AND
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The strikingly successful end-results following cooperative management of cases of toxic goiter, of complicated cases of peptic ulcer and of other types of diseases requiring specialized preoperative and postoperative attention find a decidedly suggestive parallel in the group management of surgical diseases of the colon. The combination of resources made available by isolation of patients with surgical lesions of the colon into a single division under the combined management of surgeon and clinician has brought results as striking, in our experience, in the short length of time in which it has been employed, as the results obtained from any segregated group of unusual cases. Not only has isolation of these patients afforded individualization, with better selection of cases for different types of operation, but collective management in the postoperative period has been equally invaluable. Colonic conditions are complicated; they extend over a long period before the patients who suffer from them submit to surgical procedures, and therefore colonic cases have borne not only an unjustifiable surgical mortality but an undeserved reputation for high morbidity and unfavorable prognosis. The grouping of patients with surgical conditions of the colon in a division apart from that of general surgery, and the insistence that their preoperative management be cooperative and directed along distinct lines and that the surgical management at operation be carried out by a small group of surgeons particularly interested in these diseases have, we believe, materially aided us from every standpoint.

During the six months beginning Oct. 1, 1927, and ending April 1, 1928, 147 patients with colonic disease for whom surgical treatment has been prescribed have been isolated in a single section under the medical supervision of A. H. Logan and J. Arnold Bargen, and have been operated on by Walter E. Sistrunk and Fred W. Rankin. Multiple operations were done in practically every instance in this group of cases. In 103 cases two or more operations were performed, and in seven cases as many as four operations. The cases are divided as follows:

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ninety-four of carcinoma of the rectum, rectosigmoid and sigmoid, five of carcinoma of the descending colon, three of carcinoma of the splenic flexure, five of carcinoma of the transverse colon, one large polyp of the transverse colon, four of carcinoma of the hepatic flexure, six of carcinoma of the cecum, one of carcinoma of the jejunum, three of diverticulitis of the sigmoid with abscess, ten of inflammatory cecal and pericecal lesions, seven of repair of postoperative fistula of the colon, four of resection for rectal stricture, two of closure of colostomy opening made elsewhere, one of resection of a portion of the ileum for obstruction with chronic ulcerative colitis and one of ileosigmoidostomy for a suspected malignant condition following healing chronic ulcerative colitis.

Of the 147 patients operated on, fourteen died, a total mortality of 9.6 per cent. There was only one death from preliminary colostomy in 108 operations; this occurred in a case of inoperable carcinoma of the rectum with obstruction. In the ninety-four cases of carcinoma of the rectum, rectosigmoid and sigmoid there were six deaths following resection, a mortality of 6.47 per cent. Of the fourteen deaths, eight occurred in cases which were pronounced inoperable at the time of exploration.

The preliminary preparation, the change in type of anesthesia and the coordinated postoperative management have resulted in lowering the number of postoperative complications, in the diminution in the number of fatal operative cases and in the application of more radical surgical procedures to the group. One of the most important factors in the treatment in colonic cases is preoperative preparation of the patient by the introduction of rehabilitation measures in addition to the application of local medicaments to the bowel to reduce the virulence of the organisms present and to overcome any obstruction which may have taken place. During the period of preoperative preparation, thorough cleansing of the bowel and filling the patient with foods which have a minimum of residue and may be stored for reserve is essential. The diet consists of a basic allowance of 2,000 calories of carbohydrate, including principally candy and fruit juices. To this, if the patients tolerate it, and the majority do, is added 50 per cent more carbohydrate, making a total of 3,000 calories daily. At ordinary rest, a patient will gain weight on such a diet; yet the bowel will remain comparatively empty. A daily urinalysis is made to note any overflow of the sugar threshold, and the results are checked by a determination of blood sugar which is made at the time of admission and again after a few days on the diet. Sugar rarely will appear in the urine, although there is usually an elevation of the blood sugar within normal limits. Many of these patients are in the later years of life, when tolerance for carbohydrate is more or less reduced; for these patients we have the basic

diet of 2,000 calories of carbohydrate to which is added a variable amount of protein or fat of low residue, or a combination of both. For convenience in calculation we add 25 per cent of fat or 25 per cent of protein, or 50 per cent of either, according to the patient's tolerance. Although the diet is an important factor, both for attaining cleanliness of the bowel and for giving the patient a fighting reserve, other means of emptying the colon have been found expedient. Depending on the amount of obstruction present, castor oil in variable doses of from 30 to 60 cc. is given by mouth in some cases. Twelve hours later, cleansing enemas of a physiologic solution of sodium chloride are given, which are repeated every twelve hours until twenty-four hours before operation. Such preparation takes from three to five days, depending on the amount of obstruction produced by the lesion, its situation in the colon and the general condition of the patient.

If barium has been used in the diagnosis of the lesion of the colon, it must be removed thoroughly. In the diagnosis of lesions of the colon by means of the x-rays, it is more important whenever possible to use thin barium and to wash it out thoroughly by enemas as soon as the roentgen examination has been made. A flat roentgen picture of the abdomen made from twenty-four to forty-eight hours after barium has been given eliminates the danger of sending a patient to the operating table with barium in the bowel. During the first twelve of the last twenty-four hours preceding the first operation, divided doses of camphorated tincture of opium are given by mouth. These serve to allay peristalsis and to quiet the patient in general. Patients are urged to drink much liquid up to twelve hours before operation. During the last twelve hours, the patient is allowed absolute rest; everything is done for his comfort and composure.

The day after the colostomy has been done, irrigations of the lower portion of the bowel which contains the tumor are begun. A physiologic solution of sodium chloride is used, and the irrigations are continued until forty-eight hours before the second stage, or resection. During the last forty-eight hours before the resection, aspiration of the lower segment by section is carried out both from the colostomy opening and from the rectum; it is done several times a day to prepare a dry operating field. The patient again is placed on the carbohydrate diet for twenty-four hours before the resection, and a simple cleansing enema of the portion of the bowel above the colostomy is given the night before operation to prevent gross fecal soiling at the time of operation. Before discontinuing irrigations of the lower portion, the rectum is examined digitally to determine whether or not it is empty.

The patient is observed carefully during the preoperative preparation in an effort to estimate surgical risk and to avoid complications, if possible. Not only is the lesion in the bowel examined carefully by

digital, proctoscopic and, whenever advisable, by roentgenologic methods, but the heart, blood pressure and lungs are carefully examined. Electrocardiograms are made whenever they are thought to be necessary. Renal function is estimated by phenolsulphonphthalein and studies on the blood urea. In older men with hypertrophied prostates and residual urine, particularly if there are rectal lesions, lavage of the bladder is instituted in an effort to avoid an ascending pyelocystitis. A careful note is made of the quantitative output of urine, and particular mention is made of any sign of edema of the extremities. This is especially significant in checking the not infrequent development of postoperative nephrosis. If noted early, the development of this condition into a serious complication usually can be avoided.

An additional measure, which we trust will have an important bearing on the successful management of these cases in the future, is intraperitoneal vaccination against infection of the peritoneal cavity by the use of a mixed vaccine prepared after the methods of Herrmann, who has used it extensively in experimental work. Experience with this type of medication has been too short to warrant an opinion as to its value, but we hope to be able to report more favorably on it in a future communication.

Rehabilitation after resection of a malignant tumor and establishment of colostomy is an important adjunct in the management of the patient. An effort to regulate the time of fecal evacuation from the colostomy opening, as well as to aid the establishment of formed stools by making the patient somewhat constipated, is necessary. The patient is advised, therefore, to eat at regular intervals, to include constipating foods in his diet and to avoid drinking between meals. Another significant consideration is the choice of the colostomy bag. It must be as inconspicuous as possible, should not create a vacuum by its pull on the exposed part of the colostomy, must be easily cleaned and should be relatively inexpensive.

The general acceptance of colostomy for growths in the left colon is perhaps one of the greatest advances in surgery of the colon in the last two decades. Whether the colostomy should be permanent when growths in the upper part of the rectum and sigmoid may be removed safely or whether an anastomosis should be made to reestablish continuity of the bowel is still a question. Although the technical maneuver is feasible, there are surgeons who argue, with considerable logic on their side, that growths in the sigmoid should be treated like growths in the rectum, with colostomy and extirpation of the segment of bowel below it. Usually, in cases in which colostomy is employed, because of the preliminary cleansing of the bowel and irrigation of the local growth to overcome obstruction, we have been able to leave the

bowel intact until the sixth day or longer; then, as gas begins to accumulate and the patient becomes uncomfortable, puncture is made with the cautery to relieve the tension. Subsequently, after healing is insured, the bowel is divided across its entire lumen. Following each operation it has been our custom, satisfactorily carried out, to insist on abstinence from any food or drink by mouth for seventy-two hours or longer. Good evidence that the gastro-intestinal tract is functioning satisfactorily and that infection is not taking place is the patient's ability to expel flatus. After this, administration of liquids by mouth is begun, and gradually the diet is increased. Adequately prepared patients are surprisingly free from gas in the postoperative period, and it is our experience that much of the discomfort following abdominal operations, on both the colon and the viscera, is the result of feeding the patients too early in convalescence. In colonic cases, subcutaneous administration of physiologic solution of sodium chloride or of solution of dextrose (d-glucose) is begun postoperatively, and a continuous flow at the rate of 100 cc. each hour up to 2,000 cc. in twenty-four hours is administered over the necessary period of time. Once the colostomy is done, irrigations are begun through-and-through, so as to alleviate further the local condition and to assist in adequate preparation for the subsequent resection. Usually the interval between the first and second stages of the operation has been from ten to sixteen days.

The selection of the type of operation is not entirely standardized, and the type of operation varies considerably with the half of the colon which is operated on, but the institution of drainage and the principle that most resections of the colon should be graded procedures is unquestionably a step in advance. Although it has been our aim in this group of cases to select the most radical surgical procedure compatible with the general condition of the patient, in the main, graded maneuvers rather than single-stage operation have been done. The graded operation is advantageous from many standpoints and in our hands has been instrumental, we believe, in a most satisfactory lowering of mortality.

To evaluate an aseptic technic for the removal of carcinoma of the colon is difficult. It is only one and one of the least significant steps in the development of surgical treatment of malignant conditions of the gastro-intestinal tract and yet it offers many advantages, and any procedure which accomplishes removal of intestinal carcinoma without opening the intestinal lumen, and with satisfactory functional results, merits consideration. To accomplish successfully a blind resection one must minimize the danger of infection of the peritoneum, which is the main cause of failure in gastro-intestinal surgery, and many simple methods available today emphasize the ease of its accomplishment and make its allocation among other maneuvers more ready.

Each of the two segments of the colon demands a definite technical operation, even though the principle of performing removal of the carcinoma in a graded procedure is adhered to. It has been our custom to attack right-sided colonic carcinomas by first making an aseptic ileocolostomy, and then in about two weeks removing the right half of the colon and from 30 to 35 cm. of the ileum. We believe the end-to-side transplantation of the ileum into the transverse segment of the colon is the most satisfactory type of procedure in this situation, and it can be accomplished easily and with satisfactory functional results by any of the aseptic methods popular today. The dangerous part of this operation is the secondary resection. It, likewise, may be accomplished in a clean manner, but it is more likely to be followed by peritonitis because of contamination from the necessary mobilization of the infected growth.

Colocolostomy or ileocolostomy and subsequent resection of the ascending colon and a portion of the transverse colon is the operation of choice for carcinomas of the transverse colon, unless one resorts to an obstructive operation. The latter type of operation in our service has proved exceptionally useful. We believe that in the majority of cases in which there is little obstruction and in which adequate preoperative measures have been instituted to cleanse the bowel and in this manner to eliminate a certain amount of infection, an obstructive operation may be done. In these cases one may resect the transverse colon or, indeed, any other mobile segment of the bowel and may leave on a clamp much more satisfactorily than by doing the old-time exteriorization procedure of Mikulicz. For this Mikulicz procedure we heartily advocate the substitution either of this obstructive resection or of drainage and at a second stage resection and reestablishment of the continuity of the bowel. On our service, the Milkulicz procedure practically has been abandoned except in a small group of cases of elderly patients who have small, scirrhus carcinomas of mobile segments of the large bowel which readily may be brought outside the abdomen without any preliminary measures of mobilization. In obstructive resection or colostomy and subsequent resection, one avoids the disadvantages of the Mikulicz procedure, for in the former there is no question of implantation of cancer cells in the cut surfaces of the abdominal wound, and more widespread dissection of lymph nodes and removal of the tissues contiguous to the growth is made possible. In cases of carcinoma below the transverse colon and splenic flexure, colostomy and subsequent removal is most advantageous in practically every case. The question of reestablishment of the lumen of the bowel depends on the height of the growth in the left side of the colon. Below the middle of the sigmoid, there is without question a disadvantage in joining again the cut ends of the bowel.

The question of operability, likewise, is not always easy to decide. Obviously, hepatic metastasis rules out radical operation except in an occasional case in which a small growth may be resected, by a simple maneuver, to relieve the patient of a local malignant growth. This happens infrequently and, for all practical purposes, may be forgotten. However, the question of inoperability, because of local conditions attendant on attachment to surrounding structures, is frequently perplexing. If the growth is free and movable and may be removed with or without restoration of the lumen of the bowel, the problem is simple, but if the growth is attached to the parietal peritoneum, to the small bowel or, as it occasionally is, to some other viscus which may be sacrificed without too much operative risk, the question as to the advisability of double resection, or resection and some additional operative maneuver, is not easily answered. More often than not, growths which are attached to the lateral parietal peritoneum have penetrated through the peritoneum and occasionally have formed definite pockets which will be ruptured by the slightest operative manipulation to determine the mobility and fixation of the segment of bowel. Many times we have been driven to resect a growth which had become attached to the parietal peritoneum and which had penetrated, but the penetration was not recognizable until the finger slipped easily into a small pocket during the examination. Mortality is relatively high following this accidental rupture of an abscess because of the direct contamination, and one of the greatest factors in the reduction of operative mortality will be the recognition of growths which are irremovable before some such accidental discovery forces the surgeon to perform resection. Under obstructive conditions the region of the growth becomes highly infective. Infection may take place either from increased permeability of the wall of the bowel or from direct invasion of bacteria through the broken mucosa at and around the large, ulcerating, infected, penetrating and attached growth, which may or may not have an abscess form around it.

Advanced growths which have attending complications due to their fixation, immobility and perforation of the wall of the bowel demand the exercise of meticulous judgment to decide not only whether they are amenable to radical resection but also whether palliative operation is indicated. Fixation in carcinoma of the colon, in the vast majority of instances, is due to inflammatory reactions rather than to direct invasion. The graded operation permits recession of this inflammatory reaction in many instances, with subsequent lessening of the fixation. There is then the likelihood that more radical procedures for removal of the local growth and its offending gland-bearing areas may be instituted with a most satisfactorily small operative mortality.

The choice of anesthetic in colonic cases is not unimportant. Miles, years ago, advocated spinal anesthesia in performing his abdomino-

perineal resection for carcinoma of the rectum, but it has not been universally adopted because of the dislike of general surgeons, as a class, for spinal anesthetization. In our service, during the last two years, we have employed spinal anesthesia as a routine for colostomy and the subsequent resection of all carcinomas of the rectum and for the drainage operation and removal of resectable carcinomas of the colon. The advantages of this procedure have been manifold, and we are convinced that it has had a satisfactory effect on the statistics of immediate operative mortality. Most infrequently we have found it necessary to complement the spinal anesthesia with nitrous oxide, oxygen or ethylene. The only contraindication to the use of spinal anesthesia, to which we have adhered, has been a marked lowering of the systolic blood pressure. The blood pressure is sustained throughout the operation by the administration of ephedrine. Despite an occasional reaction while the patient is on the operating table, we have no inclination to discontinue the use of spinal anesthesia. We feel confident that although the margin of safety may be smaller than that in the routine administration of anesthesia by inhalation, the freedom from pulmonary complications and the relaxation and additional ease in operating warrant the employment of this procedure in this type of case. The operative mortality is necessarily higher in this group than it is in other types of cases, and the risk justifies employment of more radical measures if they are of even slight advantage.

A REVIEW OF ABDOMINAL SURGERY*

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GALLBLADDER

Moynihan[1] found that infection began in the outer coats of the gallbladder in 63 of 81 cases of cholecystitis. In 18, the inner coats were primarily involved. Infection may reach the outer coats by direct extension from the liver, by means of the lymphatics communicating directly between the liver and gallbladder or by involvement of the peritoneal surface as a consequence of disease in an adjacent organ. In cases of infection from within, the ascending route is followed more frequently than is realized. Cholecystitis as a rule is only a part of an infection which has its origin elsewhere. Hepatitis is commonly, if not always, present, and is almost certainly of earlier origin than the inflammation of the gallbladder. Moynihan stated that sufficient regard has not been paid to the spleen as an infective agent. In some cases with recurrent stones and with marked infection in the common bile duct, the spleen may be a causative factor, and splenectomy in such cases may be indicated.

Moynihan stated that the presence of all or most of the symptoms of cholecystitis over a period of weeks or months is sufficient indication for operation. The symptoms, he emphasized, are flatulence and fulness after meals, epigastric discomfort, pain, sudden nausea, acidity, waterbrash and sallow complexion. The Graham-Cole procedure is a valuable diagnostic aid, because the concentration of bile has been shown to be checked at times when the external appearance of the viscus is only slightly altered. If inaugural symptoms are present and the cholecystographic shadow is absent or delayed, operation may be performed.

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1. Moynihan, Berkeley: Mitchell Banks Memorial Lecture on the Gall-Bladder and Its Infections, Brit. M. J. **1**:1, 1928.

Moynihan many times has hesitated to take the gallbladder out in cases of inveterate mild dyspepsia; when he has not done so, he has been compelled to operate later and has had good results. He was of the opinion that the gallbladder should be removed more frequently than is now being done.

Wilkie [2] of Edinburgh also stressed the importance of early surgical intervention in disease of the gallbladder. In cases of flatulent dyspepsia, in which the gallbladder seems implicated, he advised removal.

[COMPILERS' NOTE.—Recent studies of the functions of the gallbladder demonstrate that it is not merely a vestigial organ, and although its loss is well tolerated in man, we believe that the best indication for its removal is the clinical history and especially the presence of colic. Patients who give such a history usually will be relieved of symptoms by removal of the gallbladder even in the absence of any grossly demonstrable pathologic lesion. Patients with symptoms of mild dyspepsia and indefinite complaints relative to the upper part of the abdomen are not always cured by removal of a gallbladder which appears normal. The Graham-Cole procedure is a good diagnostic aid, but it should not be used as an indication for cholecystectomy in the absence of a good clinical history of disease of the gallbladder. We believe, as Moynihan and Wilkie have indicated, that the best results are obtained by operating as soon as the diagnosis is made.]

Wilkie,[3] in an experimental study, concluded that infection in the gallbladder precedes the common hepatic changes seen in cholecystitis. He interposed omentum between the liver and gallbladder in rabbits and produced cholecystitis by injecting streptococci obtained from cystic lymph glands of man which drained diseased gallbladders. In cases in which the omentum had been interposed between the gallbladder and liver, the liver remained grossly and microscopically normal. In the control animals, definite localized hepatitis was produced.

[COMPILERS' NOTE.—More or less well defined localized hepatitis is such a constant occurrence in cholecystitis that its presence may be used as an indication of disease of the gallbladder even in the absence of marked pathologic changes in the gallbladder itself. We are, nevertheless, inclined to the view that the primary lesion is most often in the gallbladder, and that the localized hepatitis is secondary.]

Illingworth,[4] in 100 cases in which operation had been done, noted the frequency of intramural infection of the gallbladder and that sterile bile rather commonly is found. He concluded that cholecystitis is a

2. Wilkie, D. P. D.: Personal communication.

3. Wilkie, A. L.: The Significance of Hepatitis in Relation to Cholecystitis: An Experimental Study, Brit. J. Surg. **16**:214, 1928.

4. Illingworth, C. F. W.: Types of Gall-Bladder Infection: A Study of 100 Operated Cases, Brit. J. Surg. **15**:221, 1927.

blood-borne infection and that ascending infection, or infection from the liver, is the exception rather than the rule.

Bacteriology.—Wilkie [5] concluded that cholecystitis is a blood-borne streptococcic infection of the walls of the gallbladder. He made cultures of the whole thickness of the wall of the gallbladder and found that growth did not result as a rule, whereas cultures from the submucosa and outer coats, leaving the mucosa intact, gave growths of streptococci in 42 per cent of cases. Bile inhibited the growth of streptococci. The cystic lymph node in cholecystitis gave a growth of streptococci in 86 per cent of cases.

In only 6 per cent of cases was *Bacillus coli* recovered from bile; in only one case was it recovered from the cystic lymph node and in this case probably there was contamination from bile.

A suspension of streptococci in physiologic solution of sodium chloride was injected into the lumen of the gallbladder in rabbits and did not produce change. However, intramural injection of the suspension produced progressive chronic cholecystitis, and the organism readily was recovered. Intramural injection of the suspension of streptococci, after the cyst duct was ligated, produced chronic empyema with marked intramural changes. Calculi were produced in both types of intramural injection. Repeated intravenous injections of the streptococci gave progressive chronic cholecystitis with formation of cholesterol stones. Intramural infections of the gallbladder without ligation of the cystic duct produced only stones of cholesterol, but when the cystic duct was ligated, the stones produced were of calcium and cholesterol. The intramural injections gave pathologic changes which resembled in every way those found in diseased gallbladders in man.

Wilkie [2] emphasized the importance in making bacteriologic studies in disease of the gallbladder of making cultures from the cystic lymph node at the neck of the gallbladder. In 100 cases of disease of the gallbladder including early cases with practically no change in the appearance of the gallbladder, 84 cystic lymph nodes gave a pure culture of streptococci.

[Compilers' Note.—One cannot draw definite conclusions as to the rôle played by bacteria in producing cholecystitis by bacteriologic studies of the gallbladder and bile. Judd, Mentzer and Parkhill,[6] in 200 cases, obtained 14 per cent of positive cultures from the bile of the gallbladder. Positive cultures were most frequently obtained from light colored, grainy, purulent or blood-tinged bile. Bile of normal appearance,

5. Wilkie, A. L.: The Bacteriology of Cholecystitis: A Clinical and Experimental Study, Brit. J. Surg. **15**:450, 1928.

6. Judd, E. S.; Mentzer, S. H., and Parkhill, Edith: A Bacteriologic Study of Gallbladders Removed at Operation, Am. J. M. Sc. **173**:16, 1927.

including concentrated dark bile, was usually negative. Forty-nine per cent of positive cultures were obtained from the wall of the gallbladder and 31 per cent from gallstones. Rosenow, many years ago, obtained a much higher percentage of positive cultures. This may be due to the fact that chronically diseased gallbladders were not removed as frequently then as now.]

Etiology.—Sherwood [7] reviewed a series of 200 consecutive cases of diseases of the biliary tract occurring in the last four years. The pathologic material included other cases in the same hospital studied by Denton. He examined 400 gallbladders and characterized the lesions of the biliary tract as primarily mechanical and circulatory rather than infectious disturbances.

In Sherwood's experience, by far the greater number of disturbances of the biliary tract which may be treated successfully by the surgeon, with the greatest relief and satisfaction to the patient, are those which have their basis in obstruction to the normal flow of bile through the system of ducts. The most common cause of obstruction is the presence of stones. Stones in the extrahepatic ducts are the most serious, the gravity of the condition depending on the completeness of the occlusion. The presence of stones in the gallbladder may or may not be accompanied by symptoms or appreciable pathologic changes. Such stones often appear to be harmless foreign bodies although invariably they are a potential source of trouble and danger. Cultures made from freshly removed gallbladders which contained stones frequently gave negative results, and sections of the walls of the gallbladder did not reveal any of the characteristic changes associated with infection. On the other hand, when pathologic change is easily recognizable, such changes appear to be the result of mechanical and circulatory disturbances. That secondary infection may and often does develop as a result of the presence of stones is not denied. Sherwood believed that a disturbance or unbalance of chemistry in the body, particularly in its relation to cholesterol and metabolism of calcium, must be a significant and primary factor in the etiology of cholelithiasis.

Patients who obtained the most permanent and complete relief following operation were those with cholelithiasis who sought relief because of repeated attacks of biliary colic or because of an acute attack characterized by severe pain in the right upper quadrant of the abdomen. At operation in these cases it was found that almost invariably they were associated with impaction of a stone in the cystic or, more rarely, in the common duct; the underlying pathologic changes were due to mechanical and circulatory disturbances rather than to infection. When infection was present, it seemed to be a complication.

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7. Sherwood, W. A.: Surgical Lesions of the Biliary Tract, Ann. Surg. 88:178, 1928.

The patients with the least satisfactory results in relief from symptoms were those without stones, who sought relief not because of severe pain or colic but because of vague digestive disturbances, and who did not have more definite pathologic changes than slight fibrosis or involution atrophy. Such changes are the same as may be seen in many other tissues and organs in patients after middle age. Sherwood expressed the belief that such gallbladders should be less frequently removed than has been the practice in many clinics, and that causes for such symptoms should be sought elsewhere. The word, cholecystitis, implies the presence of infection, and Sherwood asks if it always portrays an accurate picture of the most frequently encountered and predominant surgical lesions of the gallbladder.

[COMPILERS' NOTE.—For many years we have been under the impression that not all cases of disease of the gallbladder are due to bacterial infection. A number of years ago, while observing the effects of the intravenous injection of surgical solution of chlorinated soda (Dakin's solution) in animals, Mann was surprised to note its apparent selective affinity for the gallbladder and the production of cholecystitis indistinguishable pathologically from certain cases of chronic cholecystitis in man. This, and more recent studies by Mentzer [8] and others, have led us to believe that certain groups of diseases of the gallbladder are due to chemical or metabolic disturbances. This view is strengthened by the belief that cholecystic disease is often not a disease of a single organ but of a system and of each person.]

Lyons and Swalm [9] expressed the belief that many cases in which a positive cholecystogram is obtained by the Graham-Cole method and in which, at operation, a pathologic lesion of the gallbladder is not found, are due to an obstructive catarrhal condition of the cystic duct, which will respond to nonsurgical drainage, which will remain normal and which will give a negative cholecystogram. This is particularly true in cases of low-grade cholecystitis. There is no definite symptomatology in this form of obstruction of the cystic duct and it can be diagnosed only by studies of biliary drainage.

Diseases of the Gallbladder in the Young.—Potter [10] found that pathologic lesions in the biliary tract are much more common in young persons and children than is generally recognized. He reviewed

8. Mentzer, S. H.: Cholesterosis of the Gallbladder, Am. J. Path. **1**:383, 1925; A Clinical and Pathologic Study of Cholecystitis and Cholelithiasis, Surg. Gynec. Obst. **42**:782, 1926; The Pathogenesis of Biliary Calculi, Arch. Surg. **14**:14 (Jan.) 1927.

9. Lyons, B. B. V., and Swalm, W. A.: Obstruction of the Cystic Duct of a Catarrhal Variety, J. A. M. A. **90**:833 (March 17) 1928.

10. Potter, A. H.: Gallbladder Disease in Young Subjects, Surg. Gynec. Obst. **46**:795, 1928.

226 cases from the literature and added 4 of his own in which disease of the biliary tract existed in children up to 15 years of age. He cited 2 cases of stones in the common duct in fetal life (6 and 8 months, respectively). In the entire series, gallstones were present in 140 cases, cholecystitis with stones in 44 cases, cholecystitis without stones in 59 cases and cholecystitis with jaundice in 30 cases. Stones in the cystic duct were found 14 times; in the common duct, 13 times. Hydrops of the gallbladder was found 18 times and infection of the gallbladder 43 times. There were 2 cases of malignant disease of the gallbladder in patients aged 4 and 15 years, respectively. Acute cholecystitis is relatively common in the series.

Potter believed that etiologically, and in order of their importance as causative agents in diseases of the gallbladder, are the following: influenza, appendicitis, scarlet fever, diphtheria and infections of the digestive tract regardless of origin.

Operations.—DeCourcy [11] reported a series of 400 operations on the gallbladder in which cholecystectomy yielded better operative results than cholecystostomy. He reviewed this subject and found that, in general, recognition is given the fact that symptoms are only too likely to persist after simple drainage of the gallbladder, even if there is no actual recurrence of the disease. Furthermore, an infected gallbladder, if allowed to remain within the abdominal cavity, is a menace to the security of the biliary ducts, pancreas, liver, duodenum and organism as a whole.

[COMPILERS' NOTE.—In most surgical clinics, cholecystectomy is now performed in preference to cholecystostomy in cases of chronic cholecystitis. Concerning the treatment for acute cholecystitis, there is not very uniform agreement. We prefer, whenever possible, to let the acute condition subside and then to remove the gallbladder. Cholecystostomy is done only when some particularly difficult technical procedure presents itself or the poor condition of the patient demands the least dangerous operation. In some jaundiced patients, also, it is advisable to leave in the gallbladder, as it may be useful later if a condition arose which necessitated an anastomosis between the biliary and the intestinal tracts.]

Pribram [12] reported a procedure he had used in about 200 cases of cholelithiasis, which has been successful in the most complicated and difficult cases and which has permitted him to carry out the principle of abdominal closure without drainage. The reasons which usually have

11. DeCourcy, J. L.: Cholecystectomy or Cholecystostomy? Internat. J. Med. & Surg. **41**:426, 1928.

12. Pribram, B. O.: Mukoklase und drainagelose Gallenchirurgie, Zentralbl. f. Chir. **55**:773, 1928.

been given against closure without drainage are the danger of a ligature slipping off the cystic duct, secondary hemorrhage and drainage of bile from the hepatic surface. The last mentioned complication is the one most frequently encountered. To avoid this complication, Pribram carries out the following technic: The gallbladder is first drained of all bile by means of suction, and with scissors a longitudinal slit is made in the gallbladder from the fundus to the cystic duct; all stones are removed; the exposed cystic duct is now cut between two ligatures, the mucous surfaces of the gallbladder then are charred by means of the actual cautery and any small hepatic abscesses lying behind the gallbladder are likewise destroyed; cauterization extends through to the serosa of the gallbladder; the two cauterized flaps of the gallbladder are then inverted into the fossa of the gallbladder and the serosal surfaces of the gallbladder are sutured over the fossa.

The method is recommended for those cases of cholecystic disease in which subserous extirpation of the gallbladder is technically impossible because of inflammatory changes in the wall. Raw surfaces in the fossa of the gallbladder thus are eliminated.

Biliary-Intestinal Anastomosing Operations.—Judd and Parker [13] studied 137 consecutive cases in which anastomosis of the biliary and gastro-intestinal tracts was carried out from 1919 to 1924. There were 34 cases of carcinoma of the pancreas. Contrary to the general teaching, colic with jaundice occurred in 35.29 per cent of these cases, pain with jaundice in 23.52 per cent and colic, pain and jaundice in 2.94 per cent. Thus colic or pain, or both, with jaundice occurred in 61.75 per cent and jaundice without pain in only 38.23 per cent of cases. Intermittent jaundice occurred in 5 of the 12 cases with colic alone. Gallstones were found in only 1 case. Cholecystogastrostomy was the operation of choice in this group, and was performed in 26 of the 34 cases. The average postoperative duration of life was 7.7 months. The relief from jaundice in patients who survived operation was complete and permanent in 63.63 per cent of cases.

Carcinoma of the biliary ducts occurred 9 times in the series. The lesion was situated at the ampulla in 6 cases. Colic with jaundice occurred in 22.22 per cent, pain with jaundice in 44.44 per cent and jaundice without pain in 33.33 per cent of cases. Cholecystogastrostomy was the operation of choice. The average postoperative duration of life was 12.2 months.

There were 14 cases in which it was impossible at operation to determine if the lesion was carcinoma of the pancreas or pancreatitis.

13. Judd, E. S., and Parker, B. R.: Biliary Intestinal Anastomosis for Obstructive Jaundice: Analysis of 137 Consecutive Cases, Arch. Surg. **17**:1 (July) 1928.

In this group jaundice without pain was present in 71.42 per cent of cases. Cholecystogastrostomy was again the operation of choice. Of this group, 4 patients are alive; 2 have lived six years; 1, more than three years, and 1 for one year. Eleven cases of pancreatitis are reported. Of these, 6 patients had pain and colic, and 5 did not have pain or colic.

There were 62 cases of stenosis of the biliary ducts. All but one of these patients had been operated on previously. This patient had acute cholecystitis with stones and a dense cicatricial stricture of the duct. In some patients, the trouble appeared immediately after operation, and in others obstructive symptoms did not occur until a number of months following operation. In several cases the stricture probably was due to continuation of the inflammation of the biliary duct, obliterative cholangitis. Pain and colic were common symptoms. In only 3 cases there was no jaundice; in all the others intermittent jaundice of some degree was present. Hepaticoduodenostomy was the operation of choice. Seventeen patients are living and cured more than five years postoperatively; 16 are greatly improved from two to six years postoperatively; 9 are in only fair health; 2 have occasional attacks of pain, chills with fever and jaundice, but little dyspepsia. Partial or complete contraction of the stoma occurred in 7 of the 47 cases in which hepaticoduodenostomy had been performed for stricture. Return of symptoms occurred from one to six months postoperatively. Reconstruction over a tube was carried out in each case. Six patients survived operation; 2 are completely relieved of symptoms three and a half and four years postoperatively; 3 are much improved. One patient, who apparently was recovered from biliary obstruction, died one and a half years after operation from acute gastric obstruction.

Drainage.—Richter and Zimmerman [14] have closed the abdomen without drainage in operations on the gallbladder for over twelve years and are convinced of the safety and of the advantages gained in omitting the drain. They also close without drainage after work on the common duct and after transduodenal choledochotomy as well as after simple cholecystectomy. They usually follow this practice in acutely infected cases as well as in chronic cases and in relatively aseptic cases. They do not regard the spilling of bile or of duodenal contents in the field of operation as a contraindication to primary closure. If there is uncontrollable oozing, or if in operations on the common bile duct accurate closure of the duct is not possible or some other special contraindication exists, drains are used. Richter and Zimmerman have made primary closure in 262 operations on the biliary tract.

14. Richter, H. M., and Zimmerman, L. M.: Closure of the Abdomen Without Drainage After Operations Upon the Bile Ducts, Ann. Surg. **88**:187, 1928.

Their report is based on 418 operations done in consecutive cases of disease of the biliary tract. Cholecystectomy was done in 323 cases 65 times with additional work on the common duct and 25 times associated with some other major abdominal surgical procedure. In the 232 operations of cholecystectomy, the wound was drained in 70 and closed primarily without drainage in 253 cases. Simple cholecystectomy with primary closure was done 204 times with 2 deaths, an average mortality rate of 0.98 per cent. One death was due to acute pancreatitis, the other to pulmonary embolism. The average mortality in all the cholecystectomized cases, including those with work on the common duct, or other major operative procedures, was 3.39 per cent. The percentage of fatalities is higher in the groups in which drainage was allowed than in those in which it was not provided for; the explanation is that the most difficult cases are those in which drainage most often is required. The common bile duct was operated on 84 times in this series: in 55 cases with drainage and in 29 with primary closure. Of the group of 55 patients, 10 died, and there were 2 deaths in the 29 cases without drainage. There were 23 deaths in the entire series, an operative mortality of 5.5 per cent. Sixteen of the 23 deaths were in cases in which drainage was allowed, whereas only 7 deaths occurred in the cases without drainage. Among the cases in which drainage was not instituted there was only 1 death from peritonitis. In this case cholecystectomy and transduodenal exploration of the common bile duct was done.

The advantage of omitting the drain is that the postoperative discomforts are minimized, the incidence of infection and hernia is less and the period of convalescence is shortened. The frequent escape of bile seen after operation in which the incision is drained is not seen if the drain is omitted. Two cases are cited in which operation was done elsewhere; because of technical errors a large amount of bile accumulated in the abdominal cavity, remained some time and apparently was well tolerated.

Wangensteen,[15] as a result of an experimental and clinical study and an extensive review of the literature pertaining to the question of drainage in operations on the gallbladder, concluded that all wounds in cholecystectomy should be drained.

[COMPILERS' NOTE.—While certain rules can be laid down for either draining or not draining in surgical cases, in the final analysis the question remains one for each surgeon to decide from his own experience. We have closed the abdominal wound without drainage in a number in which cholecystectomy was performed. Each case must be decided indi-

15. Wangensteen, O. H.: Should the Gall-Bladder be Removed Without Drainage? Ann. Surg. **84**:821, 1926.

vidually, and if the drain safely can be omitted, we find that the patient has a smoother and more rapid convalescence. We always institute drainage after surgical procedures on the common bile duct.]

Anomalies.—Bower [16] reported a case of a man, aged 50, who was operated on because of severe pain in the upper right portion of the abdomen. The diagnosis was acute pancreatitis or perforated duodenal ulcer. At operation, the liver was found to be entirely without a left lobe and a gallbladder or fissure was not present; extrahepatic ducts could not be definitely demonstrated. The head of the pancreas was swollen and firm.

An abstract of previously reported cases is given, which included 13 cases of absence of the gallbladder and all ducts, 14 cases of absence of the gallbladder, cystic and common bile ducts and 31 cases of absence of the gallbladder and cystic duct. Other anomalies are also briefly discussed.

[COMPILERS' NOTE.—We have seen a case of congenital absence of the gallbladder which was discovered at necropsy in a patient who during life had not had manifestations of cholecystic disease. The common duct was only slightly larger than normal. At operation, one sometimes has to search carefully for a gallbladder which has been totally destroyed as a result of chronic inflammation. Often only a little bit of shrivelled scar tissue remains.]

Cholecystography.—Dick and Wallace [17] presented two cases with untoward reactions following the intravenous injection of sodium tetraiodophenolphthalein for cholecystography. In one, acute hemorrhagic pancreatitis followed immediately the injection of the dye. In another, jaundice developed following the use of the dye, and in a third case death resulted from the oral administration of the dye in a young jaundiced patient.

Experimentally, normal bile to which sodium tetraiodophenolphthalein was added was introduced into pancreatic ducts and it produced acute pancreatitis. From this it might be inferred that in the presence of a stone of the common bile duct a similar reflux into the pancreatic ducts of bile containing the salt might occur and cause pancreatitis. In cases of obstructive jaundice, small quantities of sodium tetraiodophenolphthalein were found in the pancreatic juice, and from this Dick and Wallace concluded that injury to the pancreas is possible when the dye is used in cases of obstructive jaundice. They believed

16. Bower, J. O.: Congenital Absence of the Gall-Bladder, Ann. Surg. **88**:80, 1928.

17. Dick, B. M., and Wallace, V. G. H.: Cholecystography: Toxic Effects of the Dyes: A Clinical and Experimental Study, Brit. J. Surg. **15**:360, 1927-1928.

that in the presence of the hepatic injury that is found in obstructive jaundice, the dye is more toxic in its effect than when the livers are not so injured.

[COMPILERS' NOTE.—Mild untoward reactions such as nausea, vomiting and purging occasionally follow the administration of the salts of bromine and iodine used in cholecystography. Severe reactions fortunately are rare. Kirklin and Kendall [18] have recently prepared a new compound, di-iodo-di-ethyl-ether of di-salicylphthalein, which is administered orally in the form of a 10 per cent aqueous solution of its sodium salt. The drug is less toxic than those in general use.]

FALCIFORM LIGAMENT

Schmieden and Peiper [19] studied the development, comparative anatomy, histology and pathology of the ligamentum teres hepatis. They found that it reaches its highest state of development in man. It is not a suspensory ligament of the liver, but together with the lateral umbilical ligaments and the fascia umbilicalis, all of which are closely interwoven in the region of the umbilicus, the falciform ligament forms a support for the anterior abdominal wall. The ligamentum teres hepatis contains a number of blood vessels and lymph channels and also a great deal of smooth muscle and apparently is capable of lengthening and shortening under nervous control, although this has not been definitely demonstrated. The authors have seen two cases in which pathologically produced shortening of the ligament caused symptoms in the upper part of the abdomen which were relieved by dividing it. They were of the opinion that this shortening is due to a chronic inflammatory process. The ligament furnishes a bridge between the retroperitoneal tissues and the anterior abdominal wall. In acute pancreatitis, it is often endematous and contains areas of fat necrosis. It is suggested that the umbilical colic in appendicitis in children may be due to spasm of the smooth muscle of the ligament.

The falciform ligament also is important as a pathway for the spread of carcinoma. Metastasis has been found in the ligament in carcinoma of the stomach and liver. Together with the umbilical ligaments, the falciform ligament may furnish the route by which carcinoma of the rectum metastasizes to the liver. In primary malignant tumors of the umbilicus, as much of the ligament should be excised as possible. The vessels of the ligamentum teres hepatis form an anastomosis with those of the abdominal wall on one side and with the portal

18. Kirklin, B. R., and Kendall, E. C.: New Iodine Compound for Cholecystography, Radiology **9**:205, 1927.

19. Schmieden, Viktor, and Peiper, Herbert: Die Chirurgie des Ligamentum teres hepatis, Arch. f. klin. Chir. **152**:393, 1928.

vein on the other. This is of significance in cirrhosis of the liver, and Talma's operation, therefore, should be done not above but below the umbilicus in order to avoid interfering with the natural compensatory circulation furnished by the ligamentum teres hepatis.

[COMPILERS' NOTE.—The falciform ligament has wide normal variations in form, structure and vascularity. We never have hesitated to divide the ligament when doing so would give better exposure of the field of operation. The advantages of dividing it are particularly evident in operations on the biliary tract. We have always been careful to reunite the cut ends of the ligament before closing the abdominal wound and are not aware of any ill effects resulting from this procedure.]

PANCREAS

Schmieden and Sebening [20] reviewed a series of 1,510 cases of acute pancreatic necrosis occurring since 1918. In this series, 980 cases were in women and 520 in men. Operation was performed on 1,278 patients. There are numerous theories as to the cause of acute pancreatitis, but diseases of the biliary tract are of greatest significance in its etiology. Of 1,278 cases in which operation was performed for acute pancreatic necrosis, there was disease of the biliary tract in 894 (69.8 per cent). In 31 cases of acute pancreatitis associated with gallstones the authors found stones in the ampulla in 7. Schmieden and Sebening reported 38 cases of pancreatitis after operations on the biliary tract, of which 24 followed cholecystectomy. A preoperative diagnosis of pancreatitis is not often made. The diastase reaction of Wohlgemuth is of diagnostic value. A correct diagnosis was made in 21.8 per cent and a probable diagnosis in 17.5 per cent of all the patients operated on. The incorrect diagnosis most frequently made was acute cholecystitis.

Early operation is imperative, as 95 per cent of patients not operated on die. In Schmieden and Sebening's series, the operative mortality was 51.2 per cent. In addition to instituting drainage, significant lesions of the biliary tract should be remedied. In the patients who recover from the operation, the prognosis is good. Of 624 patients who lived, only 26 had recurrences. There may be a temporary diminution in dextrose tolerance; diabetes followed in 18 cases of this series. In 6 cases, chronic pancreatitis developed.

Chronic pancreatitis is generally a medical problem. The condition is hard to recognize even with the abdomen open, as the size and consistency of the gland vary greatly under normal conditions and during different phases of digestion. It is difficult to distinguish pancreatitis from carcinoma. Of a series of 308 patients, 127 had jaundice.

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20. Schmieden and Sebening, W.: Chirurgie des Pankreas, Arch. f. klin. Chir. **148**:319, 1927.

A series of 20 cases of pancreatic stones is reported, 11 of which were removed at operation. All these patients recovered. Of the 20 cases of pancreatic stones, only 2 were demonstrated by the roentgen rays. One hundred and twenty-eight pancreatic cysts are reported, most of which were drained to the outside; a few showed anastomosis to the gallbladder or intestinal tract. Operation in traumatic injury of the pancreas aims to convey the secretions to the outside.

Coenen [21] has interested himself in a clinical investigation to determine in which cases of acute pancreatic necrosis drainage of the common bile duct, without incision and drainage of the pancreas, is indicated. Unfortunately, the anatomic and physiologic relationship of the biliary and pancreatic ducts is not such that in the majority of cases the toxic syndrome of pancreatic necrosis can be attributed to the presence of bile in the pancreas. Coenen expressed the belief that the common bile duct should not be drained primarily in every case of acute pancreatitis, since this method would bring relief only in those cases in which the pancreatic necrosis originated from the penetration of bile into the pancreatic system. Pancreatitis originating in other ways would not be affected by drainage of the choledochus.

In acute pancreatitis any unnecessary delay in instituting a surgical procedure may seriously handicap the chances of a patient's recovery. Free incision of the diseased pancreas and drainage with gauze strips and rubber tubing are of value in acute pancreatitis.

An absolute indication for drainage of the choledochus may be said to exist, if on examination of the biliary system an associated cholecystitis and cholelithiasis is found to be present. A relative indication for drainage of the choledochus exists if the common bile duct is dilated, or if cloudy bile is aspirated from the common bile duct in cases of pancreatic necrosis. In cases of traumatic pancreatic necrosis drainage of the common bile duct should be instituted only when the common duct is injured.

Grassberger [22] studied quantitatively the pancreatic ferments in the blood in diseases of the pancreas, paying especial attention to those patients with gastric or duodenal ulcer penetrating into the pancreas. He investigated especially the atoxyl resisting lipases which are specific to the pancreas. There is no method as yet for differentiating the diastase of the pancreas from that of the salivary glands. Grassberger showed that pancreatic ferments in the blood vary but little under normal conditions and after operations on abdominal organs other than the

21. Coenen, H.: Zur Indikationsstellung der Gallengangsdrainage bei der Pankreasnekrose, Zentralbl. f. Chir. **55**:900, 1928.

22. Grassberger, A.: Fermentuntersuchungen im Blut bei Pankreaserkrankungen mit besonderer Berücksichtigung des in das Pankreas penetrierenden Magen-Duodenalulcus, Deutsche Ztschr. f. Chir. **210**:293, 1928.

pancreas. In a series of gastric and duodenal ulcers, he was unable to determine whether or not they had penetrated into the head of the pancreas by a study of the ferments in the blood. In acute disease of the pancreas superimposed on a chronically diseased organ there is an increase in the pancreatic lipases in the blood.

Bernhard [23] was able to demonstrate hyperglycemia in pancreatic diseases. In mild cases, 50 Gm. of dextrose is given by mouth and even those patients with only slight pancreatic necrosis showed a delayed curve of blood sugar. The method is of value in distinguishing between disease of the biliary tract and pancreas in certain mild, obscure cases. The hyperglycemia is due probably to decreased production of insulin, and the administration of insulin to the patient is indicated. Bernhard was of the opinion that diseases of the pancreas are a more frequent cause of symptoms referable to the upper part of the abdomen than is generally realized.

[COMPILERS' NOTE.—The diagnosis of chronic pancreatitis, even with the abdomen open, is not always easily made, nor can its degree be accurately measured. Chronic pancreatitis does, however, occur frequently in association with chronic cholecystitis and seems to be a frequent cause of the continuation of symptoms following cholecystectomy. Patients with pancreatitis may continue to have symptoms for from several months to a year or more after operation. In giving a prognosis, it is important to bear this fact in mind.

In excising a gastric ulcer that is adherent to the pancreas, or in doing a resection for gastric ulcer or carcinoma of the stomach, we frequently have been forced to resect a small portion of the pancreas. Occasionally when this has been done a little pancreatic fluid will drain for a time. In all our cases, however, healing has been spontaneous and there have been no untoward effects.

The problem presented by acute pancreatic necrosis is by far the most serious one in diseases of the pancreas. Early operation is imperative, a procedure which sometimes is handicapped by the difficulty in making the diagnosis. Occasionally a true pancreatic abscess forms which may work its way to the anterior abdominal wall. We have seen this occur preoperatively and also following operations on the biliary tract.]

SPLEEN

Spence [24] suggested that purpura hemorrhagica is a disease of the whole reticulo-endothelial system and that three different types exist depending on the part involved and the extent of involvement. Splenec-

23. Bernhard, F.: Ueber die Hyperglykaemie bei akuten Pankreaserkrankungen, Deutsche Ztschr. f. Chir. **212**:209, 1928.

24. Spence, A. W.: The Results of Splenectomy for Purpura Haemorrhagica, Brit. J. Surg. **15**:466, 1927-1928.

tomy, it is known, brings about a cure in some types of purpura hemorrhagica, and in other types it is not of value. This may be explained by the fact that probably the underlying pathologic cause is elsewhere in the reticulo-endothelial system and not in the spleen in these cases in which splenectomy is not of benefit.

The three types described by Spence are: first, those in which the whole reticulo-endothelial system is uniformly affected. The diseased megakaryocytes produce defective platelets which are destroyed in excess by a diseased spleen as well as by the remainder of the reticulo-endothelial system. In these cases, splenectomy gives poor results. The second type are those cases in which the spleen is chiefly affected and the platelets are destroyed by the diseased spleen. Compensating for this rapid destruction of platelets, the megakaryocytes are overworked and are stimulated to produce defective platelets. When the spleen is removed, the rapid destruction of platelets ceases and the bone marrow is enabled to recover and to produce normal platelets. In this type, splenectomy is of great service. The third type are those cases in which the injury is to the bone marrow and extrasplenic reticulo-endothelial system rather than to the spleen. Obviously, splenectomy in this type is useless.

Green [25] stated that splenectomy for thrombocytopenic purpura hemorrhagica is a purely empiric procedure and is based on the knowledge that splenectomy produces an increase in the blood platelets or thrombocytes. Investigators have shown that splenectomy causes this increase of platelets immediately, and in large numbers, and that they appear in increasingly greater numbers each day, reaching their greatest height from the tenth to the fourteenth day. Following this they subside to the normal figure in from three to four weeks. Little is known of the etiology or pathology of thrombocytopenic purpura, and differentiation from other diseases of the blood is often difficult. Werlhof first separated purpura hemorrhagica from other hemorrhagic diseases in 1731.

The essential characteristics of purpura hemorrhagica are: marked decrease of platelets, normal coagulation time, delayed bleeding time, no retraction of the clot and no variation in the cellular elements of the blood. Unless these factors can be demonstrated, one should hesitate before calling a given condition purpura hemorrhagica and splenectomy should not be done.

Brill and Rosenthal first associated thrombocytopenic purpura with the reticulo-endothelial system. The cells of this system are found in the blood sinuses of the spleen, the sinuses of the lymph nodes, the

25. Green, T. M.: Splenectomy for Thrombocytopenic Purpura Hemorrhagica, Internat. J. Med. & Surg. **41**:487, 1928.

capillaries of the hepatic lobules and in the capillaries of the bone marrow. Some of these cells also are observed wandering throughout the connective tissues of the body and also, clustered about the capillaries.. These cells are known as Rouget cells and seem to exert definite control over the passage of the cellular elements of the blood through the capillary wall.

In 1916, Kaznelson advocated splenectomy for this condition on the assumption that the disease was due to excessive destruction of the blood platelets from overactivity of that portion of the reticulo-endothelial system situated within the spleen. When other portions of this system are at fault, as in Gaucher's disease, splenectomy is useless since the affected part of the reticulo-endothelial system lies within the lymph nodes and bone marrow. Focal infection has been given as a causative factor in the dysfunction of the reticulo-endothelial system. In the application of the tourniquet, petechiae do not appear in acute cases and do appear in the chronic cases. The bleeding is usually capillary and, when visible, is in the nature of oozing from the gums, the nose, the uterus, the bowel and the kidney. Treatment consists of transfusion and splenectomy, neither of which is aimed at the true etiology. Transfusions are repeated during the acute stage and splenectomy is considered only when the disease becomes chronic. Irradiation with ultraviolet rays, in the author's opinion, is not of value. Green reported a case of thrombocytopenic purpura successfully treated by repeated transfusions and splenectomy.

Anschütz [26] observed that the prospects for cure by splenectomy in cases of chronic essential thrombopenia are satisfactory in the few cases studied, especially when the blood platelets remain high. Splenectomy in cases with low platelet counts also has given satisfactory results. The operative mortality ranges from 6 to 8 per cent in the chronic cases as compared with 70 to 80 per cent in the acute cases. The rapid course of the disease makes it imperative to choose the proper period of the disease for surgical intervention. The indications for surgical intervention depend on the duration and severity of the disease. The complications following splenectomy done in acute cases do not appear following operation done during the chronic stage.

[COMPILERS' NOTE.—Our knowledge of the dyscrasias associated with splenomegaly is increasing. The value of splenectomy in some of these conditions remains problematic. Giffin [27] stated, however, that in two diseases, namely, hemolytic jaundice and purpura hemorrhagica,

26. Anschütz, W.: Ueber Milzexstirpation bei Thrombopenien mit besonderer Berücksichtigung der akuten Fälle, Beitr. z. klin. Chir. **142**:1, 1928.

27. Giffin, H. Z.: Splenectomy, In: Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1927, vol. 19, pp. 637-644.

the results following splenectomy are so uniformly good that the indication for the operation depends almost entirely on a satisfactory diagnosis being made. W. J. Mayo [28] said that it is in the three blood dyscrasias, splenic anemia, hemolytic icterus and purpura hemorrhagica, that splenectomy is seen at its best.]

STOMACH

Duodenal and Gastric Ulcers.—Deaver [29] was of the opinion that operation is preferable to medical management in chronic ulcers of the duodenum and stomach. In bleeding duodenal ulcers, excision by knife or cautery, if possible, is advisable, and this should be followed with gastroduodenostomy or gastro-enterostomy. Deaver does not advocate subtotal gastrectomy for duodenal ulcer, unless, in the case of a bleeding ulcer, it is not possible to remove all of the periulcerous exudate with the ulcer. Subtotal gastrectomy, however, is indicated in gastric ulcer with very few exceptions. He favors the Billroth II type of resection.

Lewishon [30] said that duodenal ulcers and gastric ulcers are grouped collectively under the term gastroduodenal ulcers by many European surgeons because they believe that the etiologic factors are the same in both lesions. There are three surgical methods of treating ulcers: (1) local excision with or without gastro-enterostomy, a procedure which is often impossible because of an immobile duodenum or because of the site of the ulcer; (2) sleeve resection, which may or may not result in an hour-glass contraction of the stomach, and (3) partial or subtotal gastrectomy.

Lewishon stated that subtotal gastrectomy for both gastric and duodenal ulcer is the operation of choice because in this procedure the ulcers are entirely removed; the acid-bearing portion of the stomach is removed and the gastric acidity is greatly reduced thereby. He said that the mortality is low and the number of recurrences is less than after gastro-enterostomy.

[COMPILERS' NOTE.—Duodenal ulcer and gastric ulcer are separate and distinct lesions. Their etiology is not known. Duodenal ulcer occurs ten times as frequently as gastric ulcer. A duodenal ulcer almost never becomes malignant. Gastric ulcer may be malignant from the start, or it may become malignant. Duodenal ulcer is a combined medical and surgical problem. Every gastric ulcer is a surgical problem.

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28. Mayo, W. J.: The Enlarged Spleen, In: Collected Papers of the Mayo Clinic, Philadelphia, W. B. Saunders Company, 1927, vol. 19, pp. 624-631.

29. Deaver, J. B.: Chronic Ulcer of Duodenum and Stomach, Surg. Gynec. Obst. **46**:161, 1928.

30. Lewishon, Richard: Gastroduodenal Ulcers: Partial Gastrectomy Versus Gastro-Enterostomy in Their Surgical Treatment, J. A. M. A. **89**:1649 (Nov. 12) 1927.

Whenever possible in duodenal ulcer, we excise the lesion, take out a section of the pyloric sphincter muscle and close it as in gastroduodenostomy. All gastric ulcers should be excised. This is done either by local excision combined with gastro-enterostomy or by partial gastric resection. Occasionally a gastric ulcer is so situated that it cannot be excised.]

Hemorrhage from the Stomach and Duodenum.—Balfour [31] said that massive hemorrhage in patients with duodenal and gastric ulcer rarely is fatal, and that repeated hemorrhages may or may not result in severe secondary anemia. Surgical treatment in acute, massive gastric hemorrhage is to be avoided because the mortality rate is higher than when patients are treated by medical means. Duodenal ulcer is the most common cause of hemorrhage from the stomach and duodenum. Of 1,072 patients operated on for duodenal ulcer, there was a history of gross hemorrhage in 184 (18 per cent). Blood was passed by bowel alone in 25 per cent, and was vomited alone in 27 per cent. Forty-eight per cent of patients gave a history of having vomited blood and passed it by rectum. Half of the patients had one hemorrhage only; the other half could recall two or more hemorrhages. The intervals between hemorrhages varied from one month to eighteen years. Patients with duodenitis may or may not give a history of hemorrhage. The causes of gastric and duodenal hemorrhage are not clear. Sometimes erosion of the gastroduodenal or superior pancreaticoduodenal artery seems to be the cause. In some cases gross defect cannot be found in the mucous membrane to account for the hemorrhage.

Mann, as a result of his experience in experimentally produced ulcers in dogs, was of the opinion that the hemorrhage may come from vascular buds in the granulation tissue which forms in the base of a healing ulcer or that actual mucous erosions are responsible. Such erosions often heal with great rapidity and do not leave a scar.

The treatment for duodenal ulcer associated with hemorrhage is surgical. The object is to relieve the patient of the symptoms of ulcer and to protect him against recurrence of the hemorrhage. Direct excision of the lesion, or cauterization, are the best methods of attack. Indirect operative procedures are of less value. Balfour stated that only 22 per cent of duodenal ulcers can be excised.

Balfour found that gastric ulcer is complicated by gross hemorrhage in 20 per cent of cases. Fatal hemorrhage from a gastric ulcer is rare. In bleeding gastric ulcers hematemesis occurred alone in 28 per cent, melena alone in 10 per cent, and both hematemesis and melena occurred in the same person in 62 per cent of cases. Gastric ulcers were surgi-

31. Balfour, D. C.: Management of Lesions of the Stomach and Duodenum Complicated by Hemorrhage, J. A. M. A. **89**:1656 (Nov. 12) 1927.

cally accessible in 87 per cent of cases. In gastric carcinoma, gross hemorrhage occurred in only 7.5 per cent of cases. Microscopic hemorrhage, on the other hand, is common in cases of carcinoma.

Delore and de Girardier [32] stated that the question of whether hemorrhage from gastric or duodenal ulcers belongs to the domain of the clinician or the surgeon is still a subject of debate. Cases of hemorrhage cannot be referred to as mild, moderate and severe, because alarming symptoms may arise in cases of profuse hemorrhage and yet the patient may make a rapid recovery provided hemorrhage does not recur. The difficulty and danger lie in recurrence of the bleeding. The source of the hemorrhage varies. It may come from the highly inflamed mucosa surrounding the ulcer or from erosions of smaller or larger vessels; the latter occurrence is more frequent than is generally thought. The prognosis in each case is therefore doubtful. The medical treatment consists of rest, lavage with hot water, injections of serum and blood transfusions. Why one patient may recover and another may not, under medical treatment, cannot be determined. Surgical treatment, therefore, is justified, although its seriousness is not to be minimized. If the patient comes under observation during the first hemorrhage, operation should be postponed until he has recovered from the immediate shock. If a patient comes under observation after repeated hemorrhage, the indication for operative procedure is further supported. Delore and de Girardier stated that the ideal procedure at operation is resection. Gastro-enterostomy should be limited to those cases with stenosis of the duodenum.

[COMPILERS' NOTE.—In both preoperative and postoperative bleeding from the stomach or duodenum, it is well to wait as long as possible before resorting to transfusion of blood. The sudden rise in blood pressure following transfusion frequently starts a fresh hemorrhage and may do more harm than good.]

Gastrojejunal Ulcers.—Wright [33] believed that gastrojejunal ulcers may occur after partial gastrectomy, rarely after pyloroplasty, and most often after gastro-enterostomy. He expressed the opinion that the cause of the primary ulcer is probably, also, the cause of the secondary ulcer. Gastric acidity is an important etiologic factor because the secondary ulcers form at the stoma where the acids have not yet been neutralized and because gastrojejunal ulceration occurs most commonly after gastro-enterostomy for duodenal ulcer which is accompanied by high acidity. Gastrojejunal ulcers occur less frequently following gastro-enterostomy in patients in whom the acids have been markedly

32. Delore, X., and de Girardier, J.: Au suject du traitement chirurgical des hémorragies gastro-duodénales d'orgine ulcereuse, Presse méd. **36**:1092, 1928.

33. Wright, G.: Gastro-Jejunal Ulceration, Canad. J. M. & S. **63**:8, 1928.

reduced by operation. The type of suture material used, and the use of clamps, are not of much significance in the etiology. Infection is a more significant factor, and Wright never does gastro-enterostomy in the presence of perforation, preferring merely to close the opening.

Gastrojejunal ulceration usually follows gastro-enterostomy, especially the Roux type of operation or gastro-enterostomy with entero-enterostomy. Fifty per cent of the secondary ulcerations are on the line of the anastomosis and encroach on both the stomach and the jejunum. Gastrocolic fistula, when present, usually follows posterior gastro-enterostomy. When secondary ulceration occurs after anterior gastro-enterostomy, it frequently presents itself in the anterior abdominal wall. Tenderness over the left rectus muscle, opposite or a little below the umbilicus, is an important physical sign. Roentgenographic examination may show irregularity of the stoma, and the stoma always is narrowed.

The methods of treatment have varied. The ulcer may be excised locally; the anastomosis may be excised together with the ulcer and a new anastomosis may be made; the anastomosis may be excised and the normal gastro-intestinal channel may be restored; finally partial gastrectomy may be done, which according to Wright is the operation of choice. When a jejunocolic or gastrocolic fistula is present, the organs must be separated and the opening in the colon must be closed, after which the other conditions are attended to. It is best to do a secondary operation before a colonic fistula develops.

Lewishon [80] reported 92 operations of gastro-enterostomy for ulcer of the duodenum at Mount Sinai Hospital in New York from 1915 to 1920. Of these, 47 per cent resulted in cure, 19 per cent of the patients still had slight complaints and 34 per cent developed gastrojejunal ulcers. Since November, 1922, all patients with duodenal and gastric ulcers at Mount Sinai Hospital have been treated by subtotal gastrectomy. Lewishon recommended this procedure without hesitation in all cases of gastric and duodenal ulcers.

[COMPILERS' NOTE.—The percentage of jejunal ulcers following gastro-enterostomy for duodenal ulcer is difficult to determine, but in our experience the frequency is about 3 per cent. Gastrojejunal ulceration occasionally follows partial gastrectomy for duodenal ulcer. We believe that most duodenal ulcers can be handled better by some less radical procedure than partial gastric resection. That absolutely sound principles have not been arrived at in the treatment for duodenal ulcer is shown by the different methods used in various clinics and is perhaps best exemplified by what has happened at Bier's clinic in Berlin. There change has been made from doing simple gastro-enterostomy in cases of duodenal ulcer to the radical method of resection, then back again to doing gastro-enterostomy, and at the present time a stand midway

between the two is taken. We prefer, if possible, to excise duodenal ulcers, but gastro-enterostomy has given excellent results in many cases, especially if there is pyloric obstruction.]

Starlinger [34] has shown by experiments on dogs that the mucosa of the descending portion of the duodenum has increased resistance against formation of ulcer. He does not believe that it is the neutralizing effect of bile and pancreatic secretion alone which prevents formation of ulcer in dogs after end-to-side gastroduodenostomy. In man, this operation may be considered as especially likely to prevent the formation of secondary ulcers. In this procedure, however, there is the possibility of obstruction by pressure from the superior mesenteric artery. Starlinger has tried to prevent this by a modification in technic. He mobilizes the duodenum from both sides, cuts the duodenum and reunites it by end-to-end anastomosis in front of the mesenteric artery, bringing the anastomosis through a new opening in the mesocolon near the middle colic artery. He stated that only time will determine whether this method of gastroduodenostomy modeled after the Billroth I method will be of value in the surgical management of cases of gastric and duodenal ulcer.

Wilkie [2] has recently been doing gastroduodenostomy after the manner of von Eiselsberg in cases of doudenal ulcer. He joins the anterior portion of the stomach near the pylorus to the second portion of the duodenum about opposite the ampulla of Vater. In this procedure, it is necessary to mobilize the second and third portions of the duodenum, which readily can be done by incising the peritoneum along the outer or convex border of the duodenum. Wilkie believed that this procedure offers all the advantages of gastrojejunostomy and practically eliminates the danger of the occurrence of a secondary or stomal ulcer, because the acid content of the stomach is emptied into the intestine near the same point that the alkaline secretions, bile and pancreatic juice enter the bowel.

Gastritis.—Konjetzny [35] was of the opinion that recent anatomic and clinical investigations have awakened new interest in the treatment for gastritis. He gave the following suggestions for detecting gastritis in its acute and chronic form at the time of operation. In acute gastritis, there is marked reddening of the serosa and more or less edema of the gastric wall which makes it seem plastic. The regional lymph

34. Starlinger, G.: Versuche zur Säureresistenz und Geschwürsbereitschaft des infrapapillären Duodenums, sowie zum Ausbau der terminolateralen Gastroduodenostomie im Rahmen der ersten Methode Billroths, Arch. f. klin. Chir. **149**: 593, 1928.

35. Konjetzny, G. E.: Gibt es eine Anzeige zur chirurgischen Behandlung der Gastritis? Arch. f. klin. Chir. **151**:370, 1928.

nodes are freshly enlarged and soft. There is slight edema of the gastric attachments of the mesogastrium and gastrocolic ligament.

Chronic gastritis, when associated with subacute inflammatory reactions, causes reddening of the serosa overlying the pyloric antrum. This corresponds to Schoemaker's "red stomach." There is more or less thickening of the gastric wall; the thickness increases from the antrum to the pylorus. This increased thickness of the gastric wall, as the pylorus is approached, is almost pathognomonic of chronic gastritis. There may be tumor-like thickening of the pylorus, often with pyloric obstruction which Konjetzny termed "stenosing gastritis." The thickening of the gastric wall may be nodular, a type which is called hyperplastic gastritis. Perigastritis usually is present on the posterior wall. There is chronic inflammatory enlargement of the lymph nodes along the greater and lesser curvature, most marked in the region of the antrum. Clinically, these two main types are often combined; the most marked evidence of inflammatory reaction is in the pyloric region.

The problem of gastritis is of clinical significance: first, because of the frequent recurrence of attacks of acute gastritis in cases with chronic inflammatory changes in the gastric mucosa; second, because epithelial proliferation in chronic gastritis generally is conceded to have precancerous proclivities.

Konjetzny discussed the indications for the surgical treatment in acute gastritis and presented three cases in which the clinical picture resembled that of perforated ulcer with peritoneal involvement. Severe hemorrhages may occur in gastric erosions which may develop in acute gastritis, and operation, in these instances, must depend on accurate determination of the source of the bleeding. A bleeding ulcer demands operation, but generalized bleeding from acute gastritis may tax the ingenuity of the surgeon as to whether surgical or medical management would give the best results. By gastrotomy, definite evidence may be gained of the source and nature of the bleeding. Jejunostomy has given good results when performed in cases of diffuse gastric hemorrhage. Konjetzny believed that medical treatment should be given in cases of simple acute ulcerative gastritis and that they should be considered of a surgical nature only if they fail to respond to medical management. Only those cases in which the gastritis is confined to the antrum pylori are amenable to surgical treatment. Generalized gastritis is not a surgical condition.

Pyloric stenosis and pylorospasm frequently are found associated with chronic gastritis. In most of the specimens of stomachs resected for chronic gastritis, thickening of the pyloric muscle is found. In hyperplastic chronic gastritis, or polypoid gastritis, there may be evidence of carcinomatous degeneration. Such precarcinomatous and carcinomatous areas may be present also in atrophic gastritis. The

polypoid form of gastritis usually can be diagnosed with the aid of the roentgen rays, but the diagnosis of atrophic gastritis can be made only by exclusion. In certain cases, gastroscopic examination has given the necessary evidence.

Konjetzny said that pyloroplasty has not been found satisfactory in the surgical management of acute or chronic gastritis, but has proved of value in cases of pylorospasm without definite evidence of gastritis. Polypoid gastritis presents a surgical indication for resection. The surgical indications in cases of simple atrophic gastritis are indefinite. Several cases in which resection has been performed showed beginning carcinomatous changes, and surgical intervention in this group remains an open question. Gastro-enterostomy has proved definitely harmful in cases of simple chronic gastritis.

[COMPILERS' NOTE.—Inflammatory lesions have a definite place in diseases of the stomach and duodenum. Duodenitis, either alone or in association with duodenal ulcer, is relatively common. Gastritis also occasionally occurs alone but usually is found in association with gastric or duodenal ulcer. We also have operated in several cases of supposed gastrojejunal ulceration and have found only well marked jejunitis and gastritis without true ulcer.]

Pylorospasm in Adults.—C. H. Mayo [36] said that it is probable that neither duodenal nor gastric ulcer occurs without accompanying pyloric spasm. Disease of the appendix and gallbladder tends to produce pyloric spasm. The pylorus and duodenum get their nerve supply from a large branch of the vagus that goes directly from the region of the cardia to the liver. For the last two years, in cases in which trouble in the stomach is due to reflex spasm of the pylorus, C. H. Mayo has divided the muscle of the pyloric ring at the upper border; in some cases also he has cut away the superior attachment of the first 2.5 cm. of the duodenum and the last 2.5 cm. of the pylorus in the hope of separating the branches of the vagus at this point. Because lesions at the angle of the stomach produce marked pylorospasm, C. H. Mayo recently has been dividing the tissues above the angle, cutting into the wall of the stomach so as to be sure of dividing the branches of the vagi. This slight additional procedure apparently has been of some benefit, but many further observations will have to be made before definite conclusions can be reached.

Hypertrophic Pyloric Stenosis in Infants.—Kirschner [37] reported 21 consecutive operations without mortality for pylorospasm in infants.

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36. Mayo, C. H.: Division of the Vagi for Pylorospasm, Ann. Surg. **88**:669, 1928.

37. Kirschner: Die operative Behandlung des Pylorospasmus der Säuglinge, Arch. f. klin. Chir. **152**:509, 1928.

He used an incision in the median line and divided the pyloric muscle longitudinally with a knife. He emphasized that the muscle must be completely divided. The incision should extend a little onto the stomach and onto the duodenum. Kirschner considered the operation indicated when the infant has failed to improve after from five to eight days on medical management.

Kühl,[38] in discussing Kirschner's paper, pointed out that it is incorrect to give the impression that there is no risk in operating on infants for pylorospasm. He estimated the mortality rate at about 16 per cent.

Cardiospasm.—Keller,[39] in a case in which nonoperative dilatation of the cardia was unsuccessful, made an anastomosis between the fundus of the stomach and the lower end of the esophagus. The patient was cured. The operation is not offered as a substitute for nonoperative treatment of cardiospasm when done by an expert, but in view of the fact that the mortality following nonoperative dilatation in general hands is high, this operation, according to Keller, presents a method whereby the general surgeon may handle such cases.

[COMPILERS' NOTE.—In most cases, cardiac spasm can be cured by dilatation through the esophagus. A hydrostatic dilator is used and is guided into position by means of a silk thread which previously has been swallowed. Often a single dilatation is sufficient, but sometimes the procedure has to be repeated. We [40] recently were forced to operate on a patient in whom the hydrostatic dilator could not be introduced. In this case the stomach was opened and the cardiac opening was dilated manually from below.]

Gastric Ulcer and Carcinoma.—Scott [41] was of the opinion that carcinoma does not develop from chronic gastric ulcer as often as is generally believed. He thought that many of the gastric ulcers in which there is coexisting carcinoma were primarily carcinomatous. Scott stated that there is no clinical method of distinguishing between a chronic gastric ulcer and early carcinoma with ulceration. All gastric ulcers, therefore, should be treated as though they were carcinomatous.

Walton [42] was able to establish a definite relationship between gastric ulcer and carcinoma in 25 cases from a series of 228 cases of carcinoma of the stomach in which surgical treatment was employed.

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38. Kühl, in discussion on Kirschner: Arch. f. klin. Chir. **152**:173, 1928.

39. Keller, W. L.: Operative Relief of Cardiospasm Where Dilatation Has Failed, Ann. Surg. **88**:58, 1928.

40. Nagel, G. W., and Judd, E. S.: Reports of Unusual Cases Observed in the Clinic of E. Starr Judd, S. Clin. N. Amer. **7**:1391, 1927.

41. Scott, W. J. M.: The Relationship of Carcinoma and Callous Gastric Ulcer, Surg. Gynec. Obst. **46**:199, 1928.

42. Walton, A. J.: Carcinoma of the Stomach, Lancet **2**:438, 1928.

[COMPILERS' NOTE.—Opinions differ as to the number of benign gastric ulcers which may become malignant. Moynihan [43] gave a figure as high as 21.8 per cent. Most German surgeons believe the association of malignant lesions and gastric ulcer an incidental occurrence rather than that one is secondary to the other. The significant point is that a certain number of gastric ulcers are malignant, whether they were malignant from inception or not, and gastric ulcer is, therefore, a surgical lesion.]

Carcinoma of the Stomach.—Balfour [44] advocated partial gastric resection in two stages for carcinoma of the pyloric end of the stomach with lymphatic involvement, with posterior fixation and with marked inflammatory reaction of the surrounding tissues. First, an anterior or posterior Polya type of operation, with or without entero-anastomosis, is done, and the pyloric end of the stomach is closed. In from ten to twelve days, when the condition of the patient is improved and the inflammation has had a chance to subside, the pyloric end of the stomach can be removed.

Walton [42] reviewed the pessimism in the public mind regarding the fatal issue in carcinoma. This is especially true of carcinoma of the stomach because the early diagnosis is associated with much difficulty and the late prognosis is unsatisfactory. He presented a series of 229 cases of carcinoma of the stomach in which operations were done and 1,086 cases of simple chronic ulcers in which the patients, likewise, were subjected to operation during the same period, from 1913 to 1928. In this series there was one carcinoma of the stomach for every six gastric operations. Walton presented a chart showing that the cases with ulcer during this period have increased threefold, whereas the number of carcinomas has remained constant year by year. Duodenal ulcers are included in the chart and form the largest proportion of this series. Ulcers of the lesser curvature of the stomach also have increased threefold since 1913. To explain the relative increase of benign ulcer over carcinoma, Walton suggested that many patients have been saved from carcinoma by having ulcers treated surgically during the benign stage.

Since recurrences following operations for carcinoma of the stomach are found most frequently in the liver and deep lymph nodes, Walton was of the opinion that improvement in late results does not lie in more extensive operations. The fact that only about a third of the total number of patients with carcinoma are operated on when first seen led Walton to state that any persistent dyspepsia, occurring after the

43. Moynihan, B.: Personal communication.

44. Balfour, D. C.: A Method of Carrying Out Two-Stage Operations for Carcinoma of the Stomach, J. A. M. A. **90**:1936 (June 16) 1928.

age of 40 years in a patient with a clear history, is to be regarded as due to carcinoma until the disease can be definitely eliminated.

Benign Gastric Neoplasms.—Balfour [31] said that only 1 per cent of gastric neoplasms are benign. He classified 50 benign neoplasms of the stomach as follows: fibro-adenomatous polyps, 14; adenomas, 4; fibromas, myomas, fibromyomas, adenomyomas and myxofibromas, 23; hemangiomas, 4; polyposis, 4; hypertrophied mucosa, 2; papilloma, 1; dermoid cysts, 3, and hypertrophied pyloric muscle, 3.

Balfour stated that if a lesion can be shown to be present in the stomach, even in the absence of hemorrhage or marked symptoms, the treatment is surgical because it has been shown that 3 of 4 intragastric lesions are malignant (McVicar).

APPENDIX

Acute Appendicitis.—Eliason and Ferguson [45] have called attention to the fact that appendicitis has been little discussed since 1915. They found that the mortality from acute appendicitis has risen in the last twelve years. Statistics show that death occurs most frequently in those cases in which the diagnosis is delayed until the disease is not any longer confined to the appendix.

Eliason and Ferguson studied 675 cases which by laboratory reports and examination of gross specimens were proved to be cases of acute appendicitis. As soon as the diagnosis was made, operation was performed. Every patient was operated on in whom rigidity overshadowed distention, providing the vascular system was still competent. Low blood pressure associated with high temperature and cold extremities was a contraindication to operation. In cases of delayed operations, the incision was made under local anesthesia and a drain was placed in the pelvis; the appendix was not removed unless it presented in the wound. Drainage was necessary in at least one of every four cases, and was most often necessary in those patients who were less than 10 or more than 40 years of age.

From a diagnostic point of view, too much stress has been placed on tenderness over McBurney's point. Of 468 patients, only 44.7 per cent had tenderness in this region. Thirty-one per cent had rectal pain and tenderness. In 16.9 per cent of patients in whom the appendix was in the pelvis, pain was felt in the epigastrium. Rigidity was absent in 34.2 per cent of the cases in which the appendix lay to the left of the cecum and over the brim of the pelvis, and was most marked in cases in which the inflammation extended by contiguity to the abdominal wall.

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45. Eliason, E. L., and Ferguson, L. K.: Mortality Factors in Acute Appendicitis, Ann. Surg. **88**:65, 1928.

Nausea and vomiting, fever and leukocytosis are not constant reliable signs. In from 60 to 70 per cent of cases, one or more symptoms were atypical; in 18.6 per cent the leukocyte count was less than 10,000.

The mortality rate in the entire series was 5.5 per cent. In the cases in which drainage was not instituted, the mortality was 1.07 per cent. Approximately 50 per cent of the patients did not reach the surgeon until the third day, and 34 per cent did not reach the surgeon until the fourth day. The most frequent complications were peritonitis and intestinal obstruction; peritonitis was the greatest factor in the mortality.

[COMPILERS' NOTE.—The problem of acute appendicitis remains one of early diagnosis and operation. This depends on the confidence of the laity in the physician and the acumen of the physician, and is not likely to undergo any immediate radical change.]

Martens [46] stated that the mortality in patients with appendicitis associated with chills is double that of similar cases without chills. All patients with appendicitis who have chills should be operated on immediately. If twenty-four hours intervened between the first chill and the time the patient is operated on, it is advisable to ligate the ileocolic vein, especially if there is inflammatory infiltration of the mesocolon. It is best also to remove the entire meso-appendix. If pus is encountered in the meso-appendix, ligation of the ileocolic vein also is indicated. If chills develop after appendectomy for acute appendicitis, or if other symptoms of pyemia develop, Martens advised that the abdomen be reopened and the ileocolic vein ligated. If chills continue to recur after an operation for acute appendicitis, careful study of the blood picture, roentgenographic examination and punctures of the liver may reveal the presence of hepatic abscess. The timely drainage of such an abscess may prevent an otherwise fatal termination.

Deaver [47] believed that the appendix deserves first place as a focus of infection, not excepting the teeth, tonsils and sinuses. He stated that duodenal ulcer, gastric ulcer and cholecystitis, and abscesses of the liver are frequently due primarily to an infected appendix. Deaver was of the opinion that when a diagnosis of appendicitis has been made in the absence of general peritonitis, appendectomy should be done at once, because he believed that expectant treatment has resulted in many complications and deaths. He placed more stress on a high proportion of polymorphonuclear leukocytes in the differentiated blood smear than on a leukocyte count.

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46. Martens, M.: Schüttelfröste und Blinddarmentzündung, München. med. Wchnschr. **75**:335, 1928.

47. Deaver, J. B.: Appendicitis, J. A. M. A. **90**:1679 (May 26) 1928.

Drainage for Appendicitis.—Weeden [48] advised the use of the Gibson-Mikulicz drain in operations for acute appendicitis. The drain consists of a square of rubber dam folded two or three times in the form of a cornucopia. The apex of this cornucopia, which forms the lowest point of the dam, is snipped off and 3.75 cm. above this, the edges of the cornucopia are cut out making a perforation of 1.25 cm. With the finger in the apex, the tampon is filled with strips of packing. When the drain is removed on the third or fourth day, a cavity about 10 cm. in diameter remains. This method affords a large amount of drainage.

Weeden reported a series of 1,588 appendectomies for acute appendicitis. In 728 (45.84 per cent), closure was without a drain and in 860 (54.16 per cent) drainage was instituted. The mortality rate in the cases in which drainage was instituted was 4.9 per cent, and in the cases in which drainage was omitted, 0.55 per cent. In Weeden's opinion, the drain is most valuable in those cases in which there is spreading peritonitis. Other advantages are that there is immediate lessening of toxemia, as shown by lowered temperature and pulse rate; the drain is easily removed and the wound is left open, therefore there is no secondary infiltration or infection of the abdominal wall; there is practically never any pocketing or formation of secondary abscesses and fecal fistulas develop in only a small number of cases (1.2 per cent); the adhesions produced, while firm enough to form a cavity for abscess, nevertheless do not give trouble and apparently disappear later. The disadvantages of the drain are that it increases the period of hospitalization; the average time, being 23.4 days as against from 17.1 to 18.6 days with other types of drains; hernia occurs in from 11.4 to 14 per cent of cases, and there is a possibility of evisceration, but this rarely occurs. Weeden believed that the advantages of the drain far outweigh its few disadvantages.

Traumatic Appendicitis.—McGeary [49] was of the opinion that the question of traumatism as a possible etiologic factor in appendicitis is of interest to the surgeon because of its medicolegal aspects. The dependent position of the appendix, its rather poor supply of blood and its bacteria-filled lumen are factors which must be considered in estimating injury to the appendix. McGeary presented data from five cases in which appendiceal symptoms and pathologic lesions in the appendix were present after traumatism of various forms. He said that if appendicitis is accepted to be the result of an accident or of trauma,

48. Weeden, W. M.: The Gibson-Mikulicz Drain in Acute Appendicitis: With Report of 1,588 Cases, Ann. Surg. **88**:76, 1928.

49. McGeary, W. C.: Traumatic Appendicitis, Internat. J. Med. & Surg. **41**:449, 1928.

there should be good corroborative evidence that there has been a more or less definite continuous train of symptoms from the date of the accident to the time of the appendiceal attack.

Appendicitis in Children.—Walton [50] said that chronic appendicitis in children under 14 years of age is rare and often is confused with a condition that really is acute. This is due to the fact that early symptoms often are overlooked or discounted in young children. A diagnosis of chronic appendicitis in adults usually means visceroptosis and operation should not be done. However, in children the diagnosis of chronic appendicitis often in reality means an acute condition, and immediate operation is indicated.

Chronic Appendicitis.—Smith [51] expressed the opinion that in the disease known as chronic appendicitis the appendix may be implicated but often is not, and its removal frequently fails to relieve symptoms. He believed that in many cases the symptoms are due to kinking and obstruction of a mobile cecum by peritoneal bands. In cases in which the cecum is dilated and thin-walled, instead of trying to plicate or suspend it, he relieves the constriction by cutting the offending peritoneal band and covers the raw surface with a free omental graft. The cecum immediately resumes its normal tone, color, thickness and peristaltic activity. In a series of 420 cases diagnosed as cases of chronic appendicitis, Smith had only 202 cases in which he believed symptoms would be relieved by appendectomy alone. In the remaining 218 cases, it was necessary to deal with other pathologic conditions encountered; in 186 of these there were definite pericolic membranes which crippled cecal peristalsis and caused cecal block.

[COMPILERS' NOTE.—The diagnosis of chronic appendicitis probably is too frequently made. A definite history of previous acute attacks is significant in making a diagnosis. In cases of indefinite abdominal symptoms all other possible causes for distress must be investigated.]

Various Positions of the Appendix.—Wakeley and Gladstone [52] analyzed 5,000 cases in which the position of the appendix was noted at operation, at necropsy and in the dissecting room. The postcecal and retrocolic positions of the appendix were those most commonly observed; the appendix was in one or the other of these positions in 64.3 per cent of cases. The pelvic or descending position occurred in 32.1 per cent of cases. Other positions of the appendix were less commonly seen.

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50. Walton, A. J.: Appendicitis in Children, Lancet **1**:445, 1928.

51. Smith, R.: Chronic Appendicitis, Ann. Surg. **88**:678, 1928.

52. Wakeley, C. P. G., and Gladstone, R. J.: The Relative Frequency of the Various Positions of the Vermiform Appendix, Lancet **1**:178, 1928.

COLON AND RECTUM

Carcinoma.—Rankin [53] emphasized the significance of adequate preparation before resection in carcinoma of the colon and rectum. This consists primarily of adequate cleansing of the gastro-intestinal tract, supportive and dietary measures, and the institution of drainage by colostomy, preliminary to subsequent resection. Rankin performed twenty-six anterior resections of the rectosigmoid and of the upper part of the rectum with only one death.

Rankin [54] reported the use of spinal anesthesia in carcinoma of the colon, with good results. Its advantages are complete relaxation during operation and the absence of postoperative pulmonary complications. Statistical studies of large groups of cases of carcinoma of the colon and rectum indicate that in a high percentage of cases there is freedom from recurrence for from three to five years or more.

Rankin and Broders [55] made a study of the factors which influence the prognosis in carcinoma of the rectum and they found that the grade of malignancy determined according to Broders' classification is of greatest significance. They found that the total of good results following operations for carcinoma of the rectum decreased in inverse proportion to the grade of malignancy, whereas the total of poor results increased in proportion to it.

Stoger [56] stated that most German surgeons favor resection in carcinoma of the rectum, with conservation of the sphincter if possible. Of 303 cases, Stoger was able to resect the growth and to save the sphincter in 76. The mortality in this group was 15.8 per cent; 44.8 per cent remained well for a period of five years or more; 8 patients were well more than ten years and 4 more than twenty years following operation. All had normal function of the rectal sphincter.

Kirschner [57] stated that the ideal surgical method for treating carcinoma of the rectum has not been determined. He believed that every case of carcinoma of the rectum should be approached by the abdominal sacral route. By opening the abdomen, metastasis, if present,

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53. Rankin, F. W.: The Technique of Anterior Resection of the Rectosigmoid, Surg. Gynec. Obst. **46**:537, 1928.

54. Rankin, F. W.: Surgical Procedures in Carcinoma of the Large Bowel, J. Michigan M. Soc. **27**:465, 1928.

55. Rankin, F. W., and Broders, A. C.: Factors Influencing Prognosis in Carcinoma of the Rectum, Surg. Gynec. Obst. **46**:660, 1928.

56. Stoger, K.: Zur resektion des Rectumkarzinoms, Deutsche Ztschr. f. Chir. **112**:232, 1928.

57. Kirschner, M.: Alte und neue Vorschläge zur Ausrottung des Mastdarmkrebses, Der Chir. **1**:1, 1928.

can be found and dissection can be carried out under direct vision with less danger of hemorrhage and infection; also, the mortality is lower in this method. Although Kirschner formerly recommended removal of the sphincter in all cases, he now recommends saving it whenever possible. The results in these cases are satisfactory. He believes an abdominal anus is always preferable to a sacral anus.

Küttner [58] reviewed a series of 1,301 cases of carcinoma of the rectum. Sixty-three per cent occurred in men and 36.7 per cent in women. The growth was close to the anus in 0.3 per cent. The prognosis in this group was poor, as early metastasis to the inguinal lymph nodes takes place. Seven and three tenths and 11.5 per cent of tumors occurred, respectively, in the pars perinealis and in the ampulla of the rectum; in these the prognosis was good. Sixty and seven-tenths per cent occurred in the ampulla proper; in this group, the prognosis was not so favorable, as the condition usually was discovered late. Eleven per cent occurred near the flexure; most of these cases are inoperable. Küttner recommended saving the sphincter whenever possible. He makes a sacral anus, as a rule, and uses the combined abdominal sacral operation only occasionally; he stated that it must first be shown that the mortality in this method is no greater and that the results are better. He reported 480 radical operations with 108 deaths, a mortality of 22.5 per cent.

In a group of 150 cases in which operation was done in the last seven years, the mortality was 16.4 per cent. Thirteen patients were operated on by the abdominal sacral route, and in this series there were 7 amputations of the rectum, with 2 deaths, a mortality of 29.5 per cent, and 6 resections with 4 deaths, a mortality of 66.6 per cent. The results following radical operation on the patients who survived operation were: patients living three years or longer, 46.5 per cent; more than five years, 32 per cent; more than eight years, 24.1 per cent, and more than ten years, 19 per cent. Of those patients who underwent palliative colostomy, 70 per cent died within the first year, 6 per cent lived three years and 2 per cent lived five years.

[COMPILERS' NOTE.—Surgical procedures in carcinoma of the colon are accompanied by a relatively high risk because of the presence of infection and because of the relatively poor supply of blood to the colon. Delayed healing following operation is due not to an actual paucity of blood supply to the colon, but to a lack of adequate anastomosing vessels. Preoperative preparation of patients is important. In cleaning out the gastro-intestinal tract, however, care must

58. Küttner, Hermann: Der Mastdarmkrebs und seine chirurgische Behandlung, Med. Klin. **25**:4, 1929.

be taken not to lower the patient's resistance by too prolonged rest in bed, by catharsis or by insufficient diet.]

Blood Supply of the Colon.—Fischer [59] described two types of small arteries that are given off from the vessel that runs longitudinally in the mesentery, close to the attachment of the bowel. The smaller vessels supply that portion of the bowel which is near its mesenteric attachment. The larger vessels encircle the bowel and supply blood to the portion opposite the mesenteric attachment. These larger vessels pass through the bases of the appendices epiploicae. When the colon is fully distended, the vessels lie flush with the surface of the bowel, but when the intestine is collapsed they form a loop extending some little distance into the appendixes. In tying off the appendixes, these vessels are likely to be injured, thus interfering with the blood supply on the contramesial side of the bowel. Fischer recommended end-to-end anastomosis of the colon, but said that the bowel should be cut through diagonally and that care should be taken not to injure the vessels already described.

Colostomy.—Demel [60] devised an artificial abdominal anus which allows the patient to control bowel movement directly. The method consists in bringing out a portion of the bowel and surrounding it with a cylinder of skin. By a special mechanical device the patient can keep this part of the bowel clamped off and thus prevent the passage of content of the bowel through the distally placed opening. Demel performed the operation on six patients with good results. Closure of the bowel is well tolerated up to twelve hours.

[COMPILERS' NOTE.—Many methods have been devised for making a colostomy which will enable the patient to control the movements of the bowel. In our experience, complicated operations are unnecessary, and properly done colostomy, with the bowel brought out through a muscle-splitting incision on the left side, gives the best results. Lavage once daily, and simple dietary measures, are adequate to enable the patient to live in comfort and to attend to his work and pleasures in the normal manner.]

Diverticulitis of the Colon.—Monsarrat [61] recommended conservative treatment rather than operation in acute diverticulitis of the colon, except in cases that have progressed to formation of abscess. Uncompli-

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59. Fischer, A. W.: Inwieweit ist durch die Ablösung des Dickdarmfettbehanges die Sicherheit der Dickdarmnaht gefährdet, Arch. f. klin. Chir. **152**:638, 1928.

60. Demel, Rudolf: Eine einfache Herstellung des verschlussfähigen Kunstafters, Deutsche Ztschr. f. Chir. **110**:336, 1928.

61. Monsarrat, K. W.: Remarks on Surgical Treatment of Diverticulitis, Brit. M. J. **2**:41, 1928.

cated cases of chronic diverticulitis, the more common form of the disease, also are best treated medically. If a patient with chronic diverticulitis is operated on, Monsarrat advised that the bowel be wrapped in omentum to prevent the formation of a fistula of the bladder.

[COMPILERS' NOTE.—A temporary colostomy by which the content of the bowel is shunted to the outside frequently is of value in diverticulitis, especially if the disease is fairly well localized. Often, after a few months, during which the lower segment of bowel is irrigated daily, the lesion has healed sufficiently to allow closure of the colostomy opening. Occasionally the lesion may be resected.]

Syphilis of the Colon.—Chastenet [62] reported two cases in which abscesses of the colon, adherent to the abdominal wall, one of which had a fecal fistula, apparently were of syphilitic origin.

Infectious Granuloma of the Colon.—Mock [63] reported three cases of infectious granuloma of the intestines. Such tumors appear to be due to excess reparative processes following low-grade infection and necrosis. The growths often reach an appreciable size and are readily mistaken for malignant tumors.

Obstruction of the Colon.—Haggard [64] stated that operations for acute intestinal obstruction are accompanied by a higher mortality than generally is supposed. The causes for this are the promiscuous use of purgatives, failure to recognize the conditions early and delay of operation. Haggard described enterostomy as a surgical method of treating intestinal obstruction. He called attention to the disturbances in chemistry of the blood and recommended the intravenous administration of solutions of dextrose and sodium chloride.

Rosenstein and Köhler [65] studied the question of the control of intestinal peristalsis in animals and showed that the injection of nicotine into the celiac ganglion caused marked increase in peristalsis in normal animals as well as in those with peritonitis. Nicotine paralyzed the synapsis between the preganglionic and postganglionic fibers of the autonomic nervous system. By injecting nicotine directly into the celiac ganglion, the action controlled by the splanchnic nerves is diminished.

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62. Chastenet, de Gery: Deux cas de sigmoïdite suppurée avec infiltration de la paroi abdominale chez des syphilitiques, Bull. et mém. Soc. Nat. de Chir. **54:** 834, 1928.

63. Mock, H. E.: Infective Granuloma of the Intestines, Internat. J. Med. & Surg. **41:**1, 1928.

64. Haggard, W. D.: Acute Intestinal Obstruction, J. A. M. A. **90:**1424 (May 15) 1928.

65. Rosenstein, Paul, and Köhler, Hans: Ueber die Beeinflussung der Darmparalyse durch Nikotininjektion in das Ganglion coeliacum, Deutsche Ztschr. f. Chir. **210:**315, 1928.

As these nerves are the antagonists of the vagi or motor nerves of the bowel, the action controlled by the latter is thereby increased. This action occurs even though the vagus is injured by toxins. The vagus is not affected by injection of the celiac ganglion, as synapsis between its preganglionic and postganglionic fibers does not occur at this point.

Following a careful study of the action of nicotine in animals, its therapeutic value was tested in human beings. The dosage used was from 5 to 8 mg. of fresh nicotine in 50 cc. of sterile water. The action of the drug appeared to take place only locally; there was no general reaction as a result of absorption of the nicotine. The 26 patients in whom it was tried all had marked paralytic ileus, mostly as a result of peritonitis, and all other methods used to relieve the ileus had failed. Rosenstein and Köhler believed that the most significant factor in causing death in ileus is the absorption of toxic products from the content of the intestine; therefore, elimination of this content is indicated. The paralysis of the bowel was overcome in 17 of the 26 patients; 9 of them subsequently died. One patient had multiple abscesses in the abdominal cavity; 2 had bronchial pneumonia; 1 died of cardiac failure; 2 died of sepsis following abortion, and 3 died as a result of the progress of carcinoma. Eight of the patients recovered.

The technic of injection is that used by Kappis and Finsterer in their method of splanchnic anesthesia; the approach is from behind, with the patient preferably in a sitting position. The method never must be used in mechanical ileus, as to use it would only make the condition worse. Rosenstein and Köhler were of the opinion that this method is a step forward in the treatment for paralytic ileus.

Ochsner, Gage and Cutting [66] induced splanchnic anesthesia in dogs with 1 per cent solution of procaine hydrochloride and found that it relieved ileus and produced peristalsis. They recommend splanchnic anesthesia in paralytic and adynamic ileus, and in mechanical ileus after the obstruction has been relieved.

PERITONITIS

Salzer [67] traced the etiology of the majority of cases of diplococcal peritonitis to infection from the genitalia. The differential diagnosis from appendicitis is possible but is necessary only in young girls. In boys there is always preceding diplococcal appendicitis which requires

66. Ochsner, Alton; Gage, I. M., and Cutting, R. A.: Treatment of Ileus by Splanchnic Anesthesia: Preliminary Report of Experimental Study, J. A. M. A. **90**:1847 (June 9) 1928.

67. Salzer, Hans: Die Diplokokkenperitonitis beim Kinder, Deutsche Ztschr. f. Chir. **208**:226, 1928.

immediate operation. Girls should be treated expectantly until the presence of pus in the abdominal cavity becomes a possibility, then operation is indicated.

David [68] showed that *Bacillus coli* was taken directly into the blood stream, as well as into the lymphatics, from the peritoneum of a normal dog. He also showed that the presence of a plastic exudate produced by intraperitoneal injection of emulsion of turpentine greatly hindered or prevented the passage of bacteria into the lymph or blood stream, whereas the presence of hypertonic solution of dextrose in the peritoneal cavity increased the rate of absorption of bacteria.

David and Sparks [69] found in an experimental study that diphtheria toxin, when injected into the normal peritoneal cavity of a dog, passed directly into the blood stream, as well as into the lymphatics, in sufficient quantities to prove fatal to guinea-pigs. In the presence of well developed plastic peritonitis, there was practically no passage of diphtheria toxin into the chyle, and it passed into the blood stream in much smaller quantities than from the normal peritoneum. The presence of hypertonic solution of dextrose in the peritoneal cavity of dogs increased the rate of absorption of diphtheria toxin by the blood stream and by the lymphatics.

David and Sparks concluded that the formation of a plastic exudate was to be regarded as a favorable process in peritonitis. Although they do not wish to press the analogy between the peritoneum of the dog and of the human being, they believed from their experiments that in the early hours of peritonitis the absorption of toxins and bacteria from the circulation directly and by way of the lymphatics is the dominant factor of danger; later, absorption from the peritoneum becomes less significant and local conditions, such as paralytic ileus, gain the ascendency.

Brown [70] found in experimental studies on cats and dogs, in which peritoneal absorption is much like that in man, that absorption by way of the thoracic duct from the peritoneal cavity is a slow and unimportant process. He also found that the omentum plays little, if any, part in actual lymphatic absorption from the peritoneal cavity. Brown concluded that lymphaticostomy cannot be recommended as a procedure offering any hope to patients suffering from acute peritonitis.

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68. David, V. C.: Peritonitis: An Experimental Study, Surg. Gynec. Obst. **45**:287, 1927.

69. David, V. C., and Sparks, J. L.: The Peritoneum as Related to Peritonitis, Ann. Surg. **88**:672, 1928.

70. Brown, K. P.: Peritoneal Lymphatic Absorption: An Experimental Investigation to Determine the Value of Lymphaticostomy, Brit. J. Surg. **15**:538, 1927-1928.

INTRA-ABDOMINAL HEMORRHAGE

Büttner [71] attempted to answer the question of whether there is any worth while method of utilizing the blood found in the abdominal cavity in patients with severe intra-abdominal hemorrhage and turning it to a useful purpose. These patients suffer from the effects of diminished blood volume and accompanying deficiency in the oxygenating properties of blood; the first requirement is to supply these deficiencies. The most satisfactory method of accomplishing this is direct transfusion of properly grouped blood. Büttner suggested, as a matter of expediency, and because grouping would not be necessary, the possibility of reinfusing the functionally active free blood found in the abdominal cavity. There is also the danger of leaving large quantities of blood in the abdominal cavity for absorption by the peritoneum. The irritated peritoneum may be protected by evacuating any extravasated blood from the abdominal cavity.

THROMBOSIS AND EMBOLISM

Borchard [72] stated that it is generally believed that thrombosis and embolism are on the increase but whether or not this is true is difficult to determine accurately. Thrombosis, embolism and infarcts must be considered together, and particular attention must be paid to the origin. Thrombosis of the saphenous vein often is incorrectly said to be the origin of a pulmonary embolus. Borchard saw a pulmonary infarct, and later thrombosis of the saphenous vein. The three factors in the causation of thrombosis are slowing of the blood stream, changes in the wall of the vessel and changes in the blood. He believed the condition of the blood to be the chief factor.

It is known that certain conditions predispose to thrombosis and embolism, namely, myoma, prostatic disease and carcinoma, especially carcinoma of the intestinal tract. On the other hand, operations on the thyroid gland are seldom followed by thrombosis and embolism. Infection increases the danger by two-thirds, especially if the infectious focus cannot be removed. Infections must also be considered which may have occurred several weeks previously, as, for example, influenza and angina. Slowing of the blood stream is significant in the first postoperative days, as the patient lies more quietly and breathes less deeply during this time. A great deal more statistical and experimental work must be done in order to clarify the ideas regarding the exact rôle of these causative factors. Treatment must be instituted largely on an empiric

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71. Büttner, Georg: Zur Therapie schwerer Blutungen in die Bauchhöhle, Arch. f. klin. Chir. **150**:93, 1928.

72. Borchard, A.: Ueber Thrombose und Embolie, Beitr. z. klin. Chir. **144**: 163, 1928.

basis. Particular attention must be paid to the patient's heart, lungs and general constitution. Age, the possible presence of endocrine disturbances and the coagulation time of the blood also must be considered.

Certain conclusions can be drawn as to the possibility of a patient developing thrombosis and embolism. Special attention should be paid to preoperative preparation. Following operation active and passive movements should be begun as early as possible. Peripheral emboli are not so much feared as formerly, as embolectomy is followed by good results. Embolus must not be considered by the surgeon as an act of fate; somewhere there must have been something which could have been foreseen or prevented.

Tempsky [73] made a statistical study of thrombosis and embolism. Only pulmonary emboli which were proved to be present at necropsy are included in the following table.

Occurrence of Emboli

	Cases	Thrombosis	Per Cent	Pulmonary Embolus	Per Cent
Carcinoma of the Stomach					
Total	1,458	45	3.8	12	0.8
Resection	496	17	3.4	5	1.2
Gastro-enterostomy	392	13	3.3	2	0.5
Exploration	407	12	2.5	3	0.6
Carcinoma of the Rectum					
Total	506	27	5.3	17	3.3
Preternatural anus	202	4	1.9	2	0.9
Amputation and resection	304	23	7.5	15	4.9
Appendicitis					
Total	1,767	29	1.6	4	0.2
Chronic	819	5	0.5		
Acute	458	7	1.5		
Perforated	490	14	2.8	4	0.8

In cases of carcinoma of the stomach, thrombosis occurred more frequently in the saphenous and femoral veins of the right side than in those of the left side. Thrombosis was never diagnosed clinically in patients of this group who died of pulmonary embolism. In the cases following appendectomy, thrombosis could not be demonstrated clinically. In one case following operation for carcinoma of the rectum, there was thrombosis of the right saphenous vein on the eighth day, and on the sixteenth day the patient died of pulmonary embolus. In appendicitis, thrombophlebitis occurred much more commonly in cases with infection, and fatal pulmonary emboli occurred only in cases with infection. Thrombosis of the extremities in this group was greater than in previous groups, and it occurred twice as often on the left side as on the right. In carcinoma of the stomach and rectum, infection does not appear to play a great rôle as a causative agent. Intravenous treat-

73. Von Tempsky, Arthur: Die Lungenembolie nach chirurgischen Eingriffen, Beitr. z. klin. Chir. **144**:170, 1928.

ment is not a cause. Cachexia and extreme amounts of fat seem to be factors in death of patients from pulmonary embolism.

Nyström [74] stated that in Sweden 5 persons have recently been operated on, after the manner of Trendelenburg, for the removal of pulmonary emboli. Three patients were operated on by Nyström, with 1 recovery and 2 deaths. The other 2 patients, operated on elsewhere, recovered. In Nyström's patient who recovered, the circulation was stopped for one hundred and four seconds while the clot was removed by means of a suction apparatus. Studies made of this patient two months after operation showed the heart and the large vessels to be anatomically and functionally normal.

Meyer [75] reported that he had seen 7 pulmonary emboli in a series of 2,182 operations in one year. Six of the patients died so suddenly that operation for emboli was not possible. In a seventh case, the operation was performed but the patient died of pneumothorax produced through a fault in technic. In another case of pulmonary embolism the operation was successful.

Giertz [76] reported from his clinic in the last six years that there were 7,824 operations and 239 cases of thrombosis or embolism. Of the cases of embolism, 27 were pulmonary and operation was done in 4 cases. Two of these patients recovered. In none of the 4 operations was any anesthetic necessary; the patient was moribund at the time of operation.

Friedel [77] reported that at the Rudolfs-Spital in Vienna occasion has arisen for doing the Trendelenburg operation 6 times. None of the patients recovered. Friedel pointed out that diagnosis is not always easy. In 2 patients who died, apparently from pulmonary embolism, necropsy revealed that death had been from cardiac causes.

Stegemann [78] examined Kirschner's patient, the first patient successfully operated on for pulmonary embolism, four years after the operation and found her normal in every way.

DeCourcy [79] stated that fully 50 per cent of the serious pulmonary complications which occur after abdominal operations are the result of embolism. Advanced age of the patient and incision through the

74. Nyström, Gunnar: Erfahrungen in drei nach Trendelenburg operierten Fällen von Lungenembolie, Arch. f. klin. Chir. **152**:450, 1928.

75. Meyer, A. W., in discussion on Nyström: Arch. f. klin. Chir. **152**:97, 1928.

76. Giertz, in discussion on Nyström: Arch. f. klin. Chir. **152**:98, 1928.

77. Friedel, Robert, in discussion on Nyström: Arch. f. klin. Chir. **152**:100, 1928.

78. Stegemann, in discussion on Nyström: Arch. f. klin. Chir. **152**:101, 1928.

79. DeCourcy, J. L.: Postoperative Embolism, Venous Stasis and Its Prevention by Proper Posture After Lower Abdominal Operations, Internat. J. Med. & Surg. **41**:179, 1928.

anterior abdominal wall are two outstanding predisposing factors in postoperative pulmonary embolism. Sepsis appears to be only a contributing factor. Rough handling of tissues, endocrine disturbances with lowered metabolism and venous stasis may be considered as other causes in the development of postoperative pulmonary embolism. The use of Fowler's position, the pillow beneath the knees after gynecologic operations and restriction of movement tend to favor massive thrombosis by retarding venous circulation. DeCourcy advocated the reverse Fowler position after surgically clean operations in the lower part of the abdomen. This position consists of elevating the foot of the bed about 15 cm. and inserting a small, fixed pillow under the small of the back.

[COMPILERS' NOTE.—Pulmonary embolus continues to be one of the most dreaded postoperative complications. Fatal pulmonary embolus fortunately is rare. Many postoperative pulmonary complications undoubtedly are embolic in origin. In our experience, fatal pulmonary embolus occurs too rapidly to consider the question of surgical intervention. The patient who survives the immediate shock often recovers under expectant treatment. The diagnosis of pulmonary embolism, while usually plain, is not always so and may be confused with certain cases of cardiac failure.]

ANESTHESIA

Wymer [80] described Ombredanne's method of ether anesthesia. A rebreathing apparatus is used in which the exhaled carbon dioxide and ether pass directly into a bag and are reinhaled. The amount of ether used can be determined accurately and is considerably less than that used in the drop method. In one case, 450 Gm. of ether was required by the open method; in a case of the same type only 150 Gm. was required by Ombredanne's method. This lessening of the amount of ether required results in an appreciable decrease in postoperative pulmonary complications.

Melzner [81] reviewed the subject of rectal anesthesia and gave his experience with tribromethanol (avertin). The manufacturers of this drug claim that it does not cause local irritation, that it does not produce unpleasant symptoms and that it is harmless and easily removed. Melzner has used the drug in 100 cases. The dosage used was 0.15 Gm. for each kilogram of body weight given as a 2.5 per cent solution in physiologic solution of sodium chloride. Morphine was given previous to injection. There was no local irritation. Light sleep was produced in from two to five minutes, and deep sleep in from ten to twenty

80. Wymer, I.: Die aethernarkose nach Ombredanne, Deutsche Ztschr. f. Chir. **212**:79, 1928.

81. Melzner, Ernst: Zur Beurteilung der Rectalnarkose mit E. 107 (Avertin), Arch. f. klin. Chir. **148**:698, 1927.

minutes following injection. Sleep occasionally lasted ten hours and longer, with no postoperative vomiting. It produced unpleasant mental disturbances in some instances. In one patient, hallucinations lasted twenty-four hours. Complete anesthesia was produced in only 54 per cent of cases and in three cases there was complete failure. Following the administration of the drug, there is an increase in the pulse rate and a fall in blood pressure; the latter, in some instances, is alarming. There were four deaths. Two occurred during anesthesia; one of these patients had a tumor of the brain; the other had a carcinoma of the stomach for which palliative gastro-enterostomy was done. One patient with exophthalmic goiter died six hours following operation and another who had undergone excision of simple goiter, died twenty-four hours postoperatively. In this case, epithelial necrosis in both kidneys was found at necropsy. Melzner advised against the use of this drug.

Martin [82] was able to produce complete narcosis in 96 per cent of cases by combining scopolamine and a proprietary double salt of morphine and narcotin with tribromethanol. The duration of full narcosis was from one and a half to two hours. The normal dosage is scopolamine, 0.00025 Gm.; proprietary double salt of morphine and narcotin, 0.03 Gm., and tribromethanol, from 0.13 to 0.15 Gm., for each kilogram of body weight. These dosages are varied slightly according to the age, weight and condition of the patient. Martin's series includes 104 cases of laparotomy.

Butzengeiger [83] has used tribromethanol in approximately 300 operations, including 200 of laparotomy, and believes that it has some distinct advantages over other forms of anesthesia. It can be given to the patient in bed, and sleep follows naturally; thereby the fear that many patients have of other forms of anesthesia is overcome. Butzengeiger did not have any fatalities. He stated, however, that at present only small doses must be tried and that complete anesthesia is not to be expected in every case.

Nordmann [84] has had favorable results with tribromethanol in 250 cases and believes that it is a step in advance in methods of anesthesia, although it is too soon to draw definite conclusions.

Sauerbruch [85] reported three cases in which tribromethanol produced ulceration of the large bowel which resulted in death; he warned against its widespread use until further careful investigation has been carried out.

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82. Martin, B.: Vollnarkosen mit Avertin, Arch. f. klin. Chir. **152**:670, 1928.

83. Butzengeiger: Die Rectalnarkose mit E. 107, Arch. f. klin. Chir. **148**: 95, 1927.

84. Nordmann, in discussion on Butzengeiger: Arch. f. klin. Chir. **148**:97, 1927.

85. Sauerbruch, in discussion on Butzengeiger: Arch. f. klin. Chir. **148**:99, 1927.

Reischauer [86] reported the case of a patient who died as a result of anesthesia with tribromethanol. Necropsy revealed marked fatty changes in the liver and kidneys.

Kirschner [87] pointed out that any new method of anesthesia must fulfil certain requirements; namely, that in all cases sufficient anesthesia must be produced and that this must be achieved without any greater risk than that which accompanies the methods of inhalation anesthesia now employed. Neither of these requirements has been fulfilled by tribromethanol up to the present time.

[COMPILERS' NOTE.—Rectal anesthesia with tribromethanol has been widely used in Germany. In a number of clinics it has been found useful and apparently in these clinics bad results have not been experienced from it. However, in an equal number, notably the clinic in Jena, Küttner's clinic in Breslau and Clairmont's clinic in Zurich, the drug is not used any longer because the physicians of these clinics believe it dangerous.]

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86. Reischauer, in discussion on Butzengeiger: Arch. f. klin. Chir **148**:100, 1927.

87. Kirschner, in discussion on Butzengeiger: Arch. f. klin. Chir. **148**:108, 1927.

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